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GASTROENTEROLOGY

ABSTRACTS AND CITATIONS

NATIONAL INSTITUTE OF ARTHRITIS, METABOLISM, AND DIGESTIVE DISEASES

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PREFACE

GASTROENTEROLOGY ABSTRACTS and CITATIONS is a publication of the National Institute of Arthritis, Metabolism and Digestive Diseases. This specialized information medium has been initiated to fill an existing great need in the field of gastroenterology and to assist the Institute in meeting its obligations to foster and support laboratory and clinical research into the nature, causes, and therapy of diseases of the gastrointestinal tract. Publication GASTROENTEROLOGY ABSTRACTS and CITATIONS makes available citations of all current papers relevant to this field from medical journals published throughout the world. Approximately one-third of the citations dealing with the major aspects of gastroenterology have accompanying abstracts.

The issuing of GASTROENTEROLOGY ABSTRACTS and CITATIONS under the auspices of the National Institute of Arthritis, Metabolism, and Digestive Diseases will provide a much needed current awareness tool to scientists and will facilitate greater integration of research and clinical efforts in this field. The number and great diversity of publications in the area of gastroenterology makes it imperative that an appropriate service be available to investigators and practitioners so that they may be apprised of progress with a minimum of delay. Our aim is to provide the readers with a readily systematized compilation of current published work. The publication will provide the greatest usefulness if these interested investigators will contribute their ideas and comments for consideration whenever possible.

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CONTENTS

	Abstracts, Citations	Page
PRE-CLINICAL SCIENCES		
Morphology, Cellular Structure and Histochemistry	5197-5236	653
Absorption--Excretion--Transport	5237-5266	659
Motility	5267-5284	665
Secretion and Metabolism	5285-5295	668
Stomach	5296-5336	670
Pancreas	5337-5352	676
Hepatobiliary Tract	5353-5560	679
Intestine	5561-5589	708
General	5590-5614	714
DIAGNOSTIC PROCEDURES	5615-5702	719
GASTROINTESTINAL DISEASES		
Esophagus	5703-5733	729
Stomach	5734-5798	731
Peptic Ulcers	5799-5859	736
Small Intestine	5860-5900	740
Malabsorption	5901-5926	743
Large Intestine	5927-6010	747
Ulcerative Colitis	6011-6023	755
Pancreas	6024-6044	757
Pancreatitis	6045-6077	760
Liver and Biliary Tract	6078-6209	765
Hyperbilirubinemic States	6210-6216	784
Nonviral Hepatitis and Hepatic Injury	6217-6233	786
Acute Viral Hepatitis	6234-6288	790
Chronic Hepatitis	6289-6310	796
Alcoholic Liver Disease	6311-6332	799
Cirrhosis	6333-6375	803
Gallbladder and Biliary Tract Diseases	6376-6475	809
General	6476-6543	822
Parasitology	6544-6576	828
Regional Enteritis	6577-6593	831
AUTHOR INDEX		
SUBJECT INDEX		

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NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *World Medical Periodicals*, 3rd Edition, are used.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	It.	Italian
Ar.	Arabic	Jap.	Japanese
Bul.	Bulgarian	Kor.	Korean
Ch.	Chinese	Latv.	Latvian
Cz.	Czech	Lith.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
E.	English	Por.	Portuguese
Eston.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fl.	Flemish	Ser.	Serbo-Croatian
Fr.	French	Sl.	Slovak
Ger.	German	Sp.	Spanish
Gr.	Greek	Sw.	Swedish
Heb.	Hebrew	Th.	Thai
Hun.	Hungarian	Turk.	Turkish
Ic.	Icelandic	Uk.	Ukrainian
In.	Indonesian	Viet.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	mm	millimeter(s)
ADP	adenosine diphosphate	mOsm	milliosmolar; milliosmeter(s)
AMP	adenosine monophosphate	msec	millisecond(s)
amp	ampere	mV	millivolt(s)
ATP	adenosine triphosphate	min	minute(s)
Å	angstrom	MTD	maximum tolerated dose
BSP	sulfobromophthalein	NAD	nicotanamide adenine dinucleotide
C	degrees centigrade	NADH	reduced nicotanamide adenine dinucleotide
cal	calories		
cc	cubic centimeter	NADP	nicotanamide adenine dinucleotidephosphate
cm	centimeter(s)		
dl	deciliter	NAPH	reduced nicotanamide adenine dinucleotidephosphate
DNA	deoxyribonucleic acid		
ED_{50}	median effective dose(s)	ng	nanogram (10^{-9})
EDTA	ethylenediamine tetraacetic acid	nm	nanometer(s)
e.g.	for example	Pa	ambient pressure
EEG	electroencephalogram	PAS	periodic acid — Schiff
ESR	erythrocyte sedimentation rate	pg	picogram (10^{-12})
g	gram(s)	pgEq	picogram equivalent(s)
µg	microgram(s)	p.o.	orally
Hz	Herz	ppb	parts per billion
hr	hour(s)	ppm	parts per million
i.m.	intramuscular	r	Roentgen
i.p.	intraperitoneal	RBC	red blood cells (erythrocytes), red blood count
IU	international unit(s)		
i.v.	intravenous	resp.	respectively
kcal	kilocalorie(s)	RNA	ribonucleic acid
kg	kilogram(s)	s.c.	subcutaneous
K_m	Michaelis constant	sec	second(s)
LD_{50}	median lethal dose(s)	SGOT	serum glutamic-oxalacetic transaminase
LDH	lactic acid dehydrogenase		
m	meter(s)	SGPT	serum glutamic-pyruvic transaminase
M	molar; mole(s)		
mM	millimolar; millimole(s)	U	unit(s)
µM	micromolar; micromole(s)	UV	ultraviolet
ma	milliampere(s)	V	volt
mCi, µCi	milli-, microcurie(s)	WBC	white blood cells (leukocytes), white blood count
mEq	milliequivalent(s)		
mg	milligram(s)	yr	year(s)
ml, µl	milli-, microliter(s)		

PRE-CLINICAL SCIENCES

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

5197 EFFECT OF TRASYLOL® ON PANETH CELLS OF THE MOUSE. *(E.)* Ahonen, A.; Penttila, A. (Dep. Anat., Univ. Helsinki, Finland). *Experientia* 31(5):577-578, 1975.

The morphological effects of Trasylol®, a trypsin and proteinase inhibitor, were studied in intestinal Paneth cells. Mice were fasted for 1 day, but received water *ad libitum*. Ten mice received an i.p. dose of 40,000 IU/kg Trasylol and seven controls received an equal dose of 0.9% saline solution. The animals were decapitated 1 hr later. Tissue samples from the mid-duodenum and mid-jejunum were formalin-fixed, paraffin-embedded, sectioned (7 μm) and stained with Best's carmine. The number of cytoplasmic coarse granules of Paneth cells was counted under a light microscope (400 cells from the experimental animals and 600 from the controls were studied). The number of identifiable Paneth cells in an intestinal crypt was higher in Trasylol-treated mice than in controls 1 hr after injection; the mean diameter of Paneth cells was also higher. There was a highly significant increase in the mean number of secretory granules/cell; from 12.1 to 17.1 in the duodenum, and from 8.3 to 19.7 in the jejunum. The inhibitory effect of Trasylol on Paneth cells is similar to the agents' effect on pancreatic acinar cells. It is suggested that the Paneth cells are morphologically, functionally and embryologically related to pancreatic acinar cells, and that the underlying mechanism for the increase of counts of secretory granules may result from delayed extrusion of the granules from the cell cytoplasm into the intestinal lumen.

5198 IDENTIFICATION OF THE INTESTINAL CELL STORING GASTRIC INHIBITORY PEPTIDE. *(E.)* Buffa, R.; Polak, J. M.; Pearse, A. G. E.; Solcia, E.; Grimelius, L.; Capella, C. (Inst. Pathol. Anat., Univ. Pavia, Italy). *Histochemistry* 43(3):249-255, 1975.

A recently-described intestinal endocrine cell was studied to determine its possible role in the production of gastric inhibitory peptide. Small intestinal mucosa samples from man, pig and dog were subjected to sequential or correlative silver impregnation techniques, applied to immunocytochemical preparations and at the ultrastructural level. The cell reacting with antigastric inhibitory peptide sera was identified as the ultrastructurally classified K cell. It was recognized by strong reactivity with the Sevier-Munger staining procedure and was chosen as the peptide-producing cell, provided that the equally strong-reacting enterochromaffin cell was excluded by virtue of its argentaffinity with the Masson technique. It is proposed that the term "gastric inhibitory peptide cell" be used to designate the K cell.

5199 INFLUENCE OF GLUCOCORTICOIDS ON SOME MORPHOLOGICAL AND BIOCHEMICAL ASPECTS OF RAT SMALL INTESTINAL MUCOSA. *(E.)* Lafont, J.; Pilon, R. (Lab. Biochim. Proteins, Univ. Sci. Tech. Languedoc, Montpellier, France). *Biochim. Biophys. Acta* 392(2):288-298, 1975.

The effect of cortisone on the rat small intestine during the post-weaning period was studied using male and female SPF rats (28-30 days old), injected with hydrocortisone (0.02, 0.1, 0.5, or 5 mg/100 g daily) i.p. or s.c. for 3 or 8 days. In another experiment, rats were injected daily with 25 μg/100 g of ACTH for 4 days. (^{3}H)thymidine was injected i.p. 0.5-12 or 24 hr before killing and cell migrations and the protein and alkaline phosphatase contents of the small intestinal mucosa were measured after sacrifice. Hydrocortisone at 5 mg/100 g for 3 days resulted in a 30% reduction in body weight and a 28% reduction in the protein content of the intestinal mucosa compared with control values. No differences were found following administration of 0.5 mg/100 g hydrocortisone for 3 days or after the administration of 0.02 or 0.1 mg hydrocortisone/100 g for 8 days. Similarly, there were no changes in body weight or intestinal mucosal protein following ACTH administration. Hydrocortisone (5 mg/100 g) caused a marked reduction in the activities of maltase and alkaline phosphatase, while ACTH elicited a specific rise in the activities of these enzymes. The opposing effects of glucocorticoid treatment and corticotrophic treatment do not appear to depend on enzyme activation, membrane stabilization, or on modifications of proliferative parameters in the intestinal epithelium. Rather, they appear to derive from the same origin, e.g., the action of glycocorticoids on the enterocyte differentiation.

5200 CHANGES IN INTESTINAL CELL KINETICS IN THE SMALL INTESTINE OF LACTATING MICE. *(E.)* Harding, J. D.; Cairnie, A. B. (Dep. Biol., Queen's Univ., Kingston, Canada). *Cell Tissue Kinet.* 8(2): 135-144, 1975.

Alterations in the proliferative characteristics of the epithelium of the mouse small intestine during lactation were studied. Virgin, pregnant (sixteenth day), lactating (fifteenth day) with 8 pups, and 15-day parous but nonlactating Swiss Webster mice were injected i.p. with ^{3}H-thymidine and, in some cases, with ^{14}C-thymidine prior to sacrifice. The number of crypts and villi in the jejunum and ileum were determined, as was the percentage of labeled mitoses. As compared with the virgin mice, the small intestine in the lactating mice was increased in size and weight. The turnover time of nonproliferating, postmitotic cells was similar in the virgin and lactating mice, as was the number of crypts. However, there was a small increase in crypt cell number in the pregnant mice and a larger increase in the lactating females (130%

increase) as compared with the virgin females. The number of crypt cells incorporating tritiated thymidine was also increased during lactation and there was evidence that the intestinal proliferating cells were cycling slightly faster in the lactating mice. The number of proliferating cells/crypt in the lactating mice was increased by 63% over the number seen in the virgin animals. Food consumption increased slightly over control levels during pregnancy and increased considerably during lactation. Lactating females in which food consumption was restricted to control levels showed increases in intestinal wet weight and intestinal crypt cell number, but the increases were smaller than those seen in lactating females fed *ad libitum*. The results suggest that food consumption, the hormonal state of the mother, and the metabolic demands of lactation all contribute to the intestinal hyperplasia seen in the lactating mouse.

5201 NEURAL STIMULATION OF MITOTIC ACTIVITY IN THE CRYPTS OF LIEBERKUHN IN RAT JEJUNUM. *(E.)* Tutton, P. J. M. (Dep. Anat., Monash Univ., Victoria, Australia). *Cell Tissue Kinet.* 8(3):259-266, 1975.

The influence of nerve stimulation and sham-stimulation on the mitotic rate of the epithelial cells lining the crypts of Lieberkühn in the jejunum of anesthetized male Sprague-Dawley rats was studied. Administration of the anesthetic (pentobarbitone sodium) and opening of the abdominal cavity were without significant effect on the crypt cell mitotic rate, which was measured 1-4 hr following the i.p. administration of cholchicine (0.1 mg/kg). Neural stimulation involved the passage of electrical current through the mesenteric nerves of an externalized loop of jejunum, while sham-stimulation involved placing the electrodes on the neurovascular bundle of the loop without passing current through them. These procedures were carried out in normal rats and in rats which had been chemically sympathectomized 3 days previously by the injection of 6-hydroxydopamine at a dose of 100 mg/kg. Externalization of a jejunal loop and application of sham-stimuli to its mesenteric nerves resulted in a significant decrease in the crypt cell mitotic rate in that loop, while the application of electrical stimuli resulted in a significant increase in the mitotic rate in the crypt cells of that segment. Similar acceleration of crypt cell proliferation by electrical stimuli applied to the mesenteric nerves was also seen in the chemically sympathectomized rats. Although the nature of the nonadrenergic nerves mediating this response is unknown at present, acetylcholinergic, serotoninergic, and/or histaminergic nerves may be involved.

5202 A REGION OF MITOCHONDRIAL DIVISION IN THE EPITHELIUM OF THE SMALL INTESTINE OF THE RAT. *(E.)* Jeynes, B. J.; Altmann, G. G. (Dep. Anat., Univ. West. Ontario, London, Canada). *Anat. Rec.* 182(3):289-296, 1975.

Ultrastructural details and mitochondrial concentrations in various regions of the small intestines of adult male Wistar rats were studied by electron microscopy. Intestinal samples were removed immediately after sacrifice at the levels of the duodenum, the jejunum and the proximal, mid, and distal ileum. Similar segments were taken from animals perfused with 3% glutaraldehyde, from animals given methotrexate (5 mg s.c. followed by 1-3 days dietary intake of 5 mg) and from animals in which the jejunum had been surgically isolated 3 weeks previously. In the control animals, the average mitochondrial number/cell section was 21 in the lower third of the crypts; it then gradually increased in the mid and upper thirds of the crypts, reaching about 42 at the villus base and remaining constant along the epithelium of the villi. The known forms of dividing mitochondria were identified in the mid and upper third of the crypts; these figures were rarely observed in the lower crypt and never seen on the villus. Crypt cells were continually produced in the lower crypt. These cells migrated to the villi while differentiating into nonproliferative absorptive cells. After methotrexate-induced mitotic inhibition, this migration continued and mitochondrial division persisted, although cell morphology was greatly changed by the third day of treatment. In segments of the jejunum isolated surgically from the functional intestine for 3 weeks, mitosis and cell migration continued, but no evidence of mitochondrial duplication was found. The results suggest that each mitochondrion probably undergoes a division as the crypt cells migrate from the mid-crypts to the villus. As a result, the villus epithelial cells contain double numbers of mitochondria. The mitochondrial division does not appear to be directly related to mitosis and is apparently elicited by a stimulus present only in the functional intestine.

5203 EARLY LYMPHOEPITHELIAL RELATIONSHIPS IN HUMAN APPENDIX: A COMBINED LIGHT- AND ELECTRON-MICROSCOPIC STUDY. *(E.)* Bockman, D. E.; Cooper, M. D. (Med. Coll. Ohio, Toledo). *Gastroenterology* 68(5):1160-1168, 1975.

The relationship between the developing lymphoid follicles and epithelium in the appendix of human fetuses and children was examined by light and electron microscopy. Appendices were obtained from nine apparently normal fetuses, one stillborn child, and five children ranging in age from 5 months to 8 yr. Follicular accumulations of lymphocytes were observed first in the mesenchyme of an 11.5-cm fetus immediately beneath the epithelium, which contained a predominance of goblet cells on the surface and in the crypts. Larger accumulations of lymphoid cells in older fetuses (15 cm and larger) were intimately related to the surface epithelium but not to the epithelium of the crypts. At the point of invasion of the lymphoid cells into the surface epithelium, the goblet cell population diminished and epithelial cells displaying a morphologically distinct form of differentiation were observed. They were characterized by the presence of irregular microvilli or microfolds and numerous apical micropinocytotic vesicles. This follicle-associated epithelium (FAE) appeared ultrastructurally identical with the epithelium in the chicken bursa of Fabricius, the murine Peyer's patch and the rabbit appendix, which has been shown

to be capable of transporting ferritin and India ink tracer from the lumen to underlying tissue. The FAE also appeared identical to the specialized epithelial cells of the adult human Peyer's patches. No FAE was observed in the youngest fetuses (11.5 cm and less), but was maintained throughout the later neonatal period into childhood. It is suggested that the FAE may provide a channel through which antigens may stimulate clonal proliferation and seeding of B-lymphocytes throughout the lamina propria of the internal mucous surfaces.

5204 CYTOCHEMICAL LOCALIZATION OF ADENYL CYCLASE IN THE RAT COLONIC MUCOSA. *(E.)* Corriveau, M.; Rojo-Ortega, J. M. (Clin. Res. Inst. Montreal, Canada). *Virchows Arch. Zellpathol.* 18(2):129-134, 1975.

The cellular distribution of adenyl cyclase was studied at the ultrastructural level in the rat colonic mucosa. Segments of the ascending and descending colonic mucosa of adult male Sprague-Dawley rats were incubated in medium containing 4 mM lead nitrate. The lead ions caused precipitation of the pyrophosphate formed by adenyl cyclase reacting with adenylyl-imidodiphosphate in the incubation medium. By this method, adenyl cyclase activity was localized in the brush border of the epithelial cells of the surface epithelium of the ascending colon. In the descending colon, coarse precipitates were located mainly in the basement membrane, in the junctional areas between epithelial cells, and on the brush border of all epithelial cells. Hydrolysis of the substrate was accelerated by the addition of 10 mM sodium fluoride to the incubation medium. The results suggest that the adenyl cyclase present in the basement membrane, plasma membrane, and in the brush borders may play a role in the regulation of intestinal transport. They also suggest that the colonic membrane may play a more crucial role in intestinal regulation than that of a simple filtration barrier.

5205 SMALL INTESTINAL MUCOSAL INJURY IN THE EXPERIMENTAL BLIND LOOP SYNDROME: LIGHT- AND ELECTRON-MICROSCOPIC AND HISTOCHEMICAL STUDIES. *(E.)* Toskes, P. P.; Giannella, R. A.; Jervis, H. R.; Rout, W. R.; Takeuchi, A. (Dep. Hematol., Walter Reed Army Inst. Res., Washington, D.C.). *Gastroenterology* 68(5):1193-1203, 1974.

The morphological integrity of the small intestine was investigated by light and electron microscopic and histochemical methods in male Walter Reed Carworth Farms rats with the experimental blind loop syndrome. Three groups of animals were studied: normal control animals and animals with either self-filling (SF) or self-emptying (SE) jejunal blind loops constructed surgically 10 cm distal to the ligament of Treitz. The blind loop segment and the jejunal segments proximal and distal to the blind loop were examined. Vitamin B_{12} malabsorption and bacterial overgrowth occurred only in the animals with SF blind loops, the most striking findings occurring in the blind loop itself with similar but less marked changes in the jejunum distal but not proximal to the blind loop segment. Hypertrophy of both crypts and villi was evident with focal abnormalities of the villus architecture, approximately 10-20% of the columnar cells in the upper half of the villi being swollen and vesiculated. By electron microscopy, the microvilli demonstrated a variety of degenerative changes and the glycocalyx and terminal web were disrupted. In addition, the mitochondrial and endoplasmic reticulum (ER), both smooth and rough, were swollen and concentric whorls of parallel membranes and long, curvilinear rough ER were present in the cytoplasm. Histochemically, there was loss of enzymatic activity in the epithelial brush border, mitochondria, and ER. Inasmuch as bacterial invasion of the jejunal mucosa was not seen, the etiology of these changes is unknown but may involve bacterial "toxins" or products of bacterial metabolism. These morphological observations demonstrate that both brush border and intracellular injury occur in the jejunal epithelial cells of rats with the experimental blind loop syndrome.

5206 FLUORESCENCE HISTOCHEMISTRY OF THE GASTRIN CELL IN FETAL AND ADULT MAN. *(E.)* Larsson, L. I.; Hakanson, R.; Sjoberg, N. O.; Sundler, F. (Dep. Histol., Univ. Lund, Sweden). *Gastroenterology* 68(5):1152-1159, 1975.

The histochemical properties of cells within the antral and duodenal mucosa and pancreatic islets which show gastrin immunoreactivity were compared in material from fetal and adult humans. Material from the adult and fetal antropyloric mucosa and proximal duodenal mucosa and adult pancreas were exposed to formaldehyde gas-ozone treatment or formaldehyde gas-hydrochloric acid treatment and examined using immunohistochemical techniques and fluorescence microscopy. Immunoreactive gastrin cells were demonstrated in both the adult and fetal duodenal mucosa and in the adult antropyloric mucosa; only a few scattered gastrin cells were detected in the antral mucosa of fetuses of 19-20 weeks gestation, but these cells were present in large numbers in the antral mucosa of two stillborn infants. No gastrin immunoreactivity was seen in the pancreatic islets. The antral and duodenal gastrin cells, as well as the pancreatic A_2-cells, displayed strong formaldehyde-ozone- and formaldehyde-HCl-induced fluorescence, whereas the pancreatic A_1-cells (previously claimed to store gastrin) were non-fluorescent. The fluorescence histochemical properties of the fetal gastrin cells did not differ from those of the adult material. The results suggest a relationship between immunoreactive gastrin and a postulated peptide having NH_2-terminal tryptophan.

5207 DIRECT EFFECTS OF ENDOTOXIN ON CANINE GASTRIC MUCOSAL PERMEABILITY AND MORPHOLOGY. *(E.)* Cheung, L. Y.; Stephenson, L. W.; Moody, F. G.; Torma, M. J.; Zalewsky, C. (Univ. Utah Med. Cent., Salt Lake City). *J. Surg. Res.* 18(4):417-425, 1975.

The direct effects of endotoxin on canine gastric mucosal permeability and morphology in the absence of arterial hypotension were evaluated. Male and female mongrel dogs were anesthetized and the fundic

section of the stomach was removed and placed in a single- or double-lumened Leucite chamber. Mucosal bathing solutions of isotonic HCl and isosmotic mannitol were used singly in the single chamber experiments and together, one in each chamber, in the double chamber experiments. *E. coli* endotoxin (0.1 ml/min) was infused continuously into the artery supplying the chambered gastric segment; during control periods, normal saline was infused. Endotoxin infusion produced a profound change in the mucosal appearance, from bright, uniform red during the control periods to mottled, pale-white during endotoxin infusion. Gross erosions occurred in 4/13 mucosae bathed with 0.15 N HCl within 1 hr of infusion of endotoxin in the absence of arterial hypotension. Histological changes seen in most experiments included a release of mucus from the surface epithelial cells and elevation of the epithelium from the basement membrane. In more advanced lesions, severe injury extended into the gastric glands and surrounding connective tissue with cellular necrosis. In spite of gross and microscopic gastric mucosal injury, no significant change was seen in hydrogen ion back diffusion or sodium efflux. These observations suggest that gastric mucosal injuries can occur in endotoxemia without systemic arterial hypotension and that anatomical mucosal injuries are not associated with the destruction of the hydrogen-sodium permeability barrier.

5208 CYTOTOXICITY OF VINBLASTINE AND VINCRISTINE TO PANCREATIC ACINAR CELLS. *(E.)* Nevalainen, T. J. (Dep. Pathol. Anat., Univ. Turku, Finland). *Virchows Arch. Zellpathol.* 18(2):119-127, 1975.

The effects of the Vinca alkaloids vinblastine and vincristine on the ultrastructural integrity of the murine exocrine pancreas cells were studied. Vincristine (20 mg/kg) or vinblastine (40 or 200 mg/kg) dissolved in saline was given as a single i.p. injection to male and female BALB/c mice; nontreated mice served as controls. The animals were killed 1, 3, 5, or 24 hr or 4 days after drug treatment and small pieces of the pancreas were prepared for electron microscopic examination. Fatty liver was found in all animals within 24 hr after treatment with either drug, and there were scattered foci of cell necrosis within 24 hr and marked loss of acinar cells and pancreatic atrophy within 4 days after drug administration. In animals killed after 1 hr, the acinar cells were slightly swollen and some areas of the rough endoplasmic reticulum were vesiculated. The number of autophagic vacuoles in the acinar cells increased progressively between 3 and 5 hr after treatment, the Golgi apparatus being hypertrophied at these times. Mitochondria and portions of the rough endoplasmic reticulum were identified within the autophagic vacuoles. The zymogen granules appeared normal but were decreased in number. These changes were more pronounced after 24 hr and 4 days, necrotic and disintegrating acinar cells being found at both times. It is concluded that the vinca alkaloids caused autophagy and degeneration in the pancreatic acinar cells and that their inhibitory effect on pancreatic enzyme secretion might depend partly or solely on their cytotoxic action.

5209 MORPHOLOGICAL EFFECTS OF INTRAPERITONEAL OILY FATTY ACIDS ON THE INTESTINE AND PERITONEUM OF RATS. *(E.)* Wagner, R.; Oehlert, W. (Dep. Pathol., Univ. Freiburg, Germany). *Beitr. Pathol.* 154(3):281-292, 1975.

The effects of the administration of oily fatty acids on the intestinal mucosa was studied in 133 rats. Sesame oil DAB 6 was injected i.p. daily in amounts of 3.3 ml/kg and 6.6 ml/kg; 43 received 3.3 ml/kg p.o. The rats were sacrificed at various time intervals, blood samples were taken, and tissues were prepared for histologic study. Other groups of rats received single i.p. injections of 5 ml, 10 ml, or 20 ml sesam oil; four rats received 3 ml daily for 5 days. They were sacrificed on various days from day 1 to day 75. No pathological changes were found in animals treated p.o. After 68-100 days of continuous daily i.p. injections, changes in the small intestinal mucosa were observed: structural villous alterations with shortening, broadening and fusion of villi, fatty vacuolization of epithelia, dilation of lymphatics and mesenchyme activation of the villous stroma with occurrence of large, PAS-positive histiocytes. The most impressive structural changes resulted from long-term low dosage or short-term high dosage of oil. The results suggest that the oil-induced granulomatous proliferative-inflammatory process of the peritoneum could be regarded as a precursor and mediator of the changes, and that absorptive overload of the peritoneum and overcharge of the mesenteric lymph drainage are pathogenic factors in the development of mucosa alteration leading to disturbed villous microcirculation and insufficient supply to the enterocytes.

5210 PERIBILIARY PORTAL SYSTEM IN THE MONKEY LIVER AS EVIDENCED BY THE INJECTION REPLICA SCANNING ELECTRON MICROSCOPE METHOD. *(E.)* Murakami, T.; Itoshima, T.; Shimada, Y. (Okayama Univ. Sch. Med., Japan). *Arch. Histol. Jpn.* 37(3): 245-260, 1974.

The peribiliary plexus and its efferent vessels in the monkey liver were studied using a method consisting of plastic casting and scanning electron microscopy combined with microdissection. The methyl methacrylate blood vascular casts of a male rhesus monkey liver and the kidney of a male crab-eating monkey were used. It was demonstrated that the peribiliary blood vascular plexus received the afferent vessels from the hepatic artery and emitted the efferent vessels directly draining into the hepatic sinusoids. The study also confirmed that the route to the sinusoids *via* the peribiliary plexus is completely isolated from the route of the portal vein as well as the arterial pathway to the sinusoids. The findings support the classical view of the "radicular portal veins" and show that the efferent vessels of the peribiliary plexus are distributed as if they cover deficiencies of the portal supply to the sinusoids.

5211 MACROMOLECULAR CONFORMATION OF THE COLON MUCUS AS REVEALED BY DETECTION OF ANISOTROPIC PHENOMENA. *(E.)* Vidal, B. C.; Mello, M. L. S. (Inst. Biol., State Univ. Campinas, Brazil). *Ann. Histochim.* 19(2):151-156, 1974.

Macromolecular components of rat colon mucus were studied after staining with toluidine blue solution (pH 4.0) and anisotropical phenomena were also investigated. The mucus was found to be metachromatic and displayed anisotropic phenomena; dichroism was visually observed, which was negative at wavelengths 490-620 nm. The anomalous dispersion of the birefringence showed an inflection point at a wavelength near that of the linear dichroism maximum; thus it was considered a Cotton effect-like phenomenon. The observations suggest a helical conformation for the acid mucopolysaccharides of the mucus and that structural conditions and/or patterns of molecular orientation of the acid mucopolysaccharides can vary in different tissues.

5212 PROLIFERATIVE PATTERNS IN THE MOUSE JEJUNAL EPITHELIUM AFTER FRACTIONATED ABDOMINAL X-IRRADIATION. *(E.)* Lesher, S.; Cooper, J.; Hagemann, R.; Lesher, J. (Allegheny Gen. Hosp., Pittsburgh, Pa.). *Curr. Top. Radiat. Res.* 10(3):229-261, 1975.

Cell proliferation was studied in the jejunal epithelium of mice exposed to fractionated abdominal x-irradiation. Lightly anesthetized male C57Bl/6J mice were exposed to x-irradiation at a dose level of 300 R; the upper portion of each animal was lead shielded. The radiation exposures were repeated 2, 4, 6, or 9 times, with intervals of 6, 12, 24, 48, or 96 hr separating the exposures. The crypt squash technique was used to measure labeled nuclei and mitotic figures/crypt 1-96 hr after the final exposure, and cell cycle times including estimates of G_1, S, G_2, and M were made 12 and 24 hr after the final exposure. The damage-repair-recovery pattern, i.e., increase in the size of the proliferative population and acceleration of the generation cycle, was similar after single (previous data) and fractionated exposures. However, there were quantitive differences between various fractionation regimens. Overshoot in labeled nuclei/crypt and mitotic figures/crypt following 2 or more exposures was less than after 1 exposure, and the G_2 block decreased as the number of fractions increased. The number of 300 R exposures which could be tolerated was dependent on the time interval between exposures. Total cell production through 9 successive exposures was estimated to be, in percent of controls, 15% (12 hr interval), 34% (24 hr interval), and 64% (48 hr interval). As a result, death followed depression of the total cell production to 15% of the control levels for 4 days (12 hr fractions), to 34% of control levels for 8 days (24 hr fractions), and to 64% of control levels for 16 days (48 hr fractions). Thus, both the degree and the duration of depressed proliferative activity are critical factors in the intestinal response to fractionated treatment. Because of its great potential for compensatory hyperplasia following injury, the intestinal epithelium is capable of recovery from extensive damage provided that critical levels of crypt depletion have not occurred and that sufficient time is allowed for the acceleration in proliferative activity. The compensatory recovery potential is maintained over a large number of exposures.

5213 STUDIES ON A FRACTION OF HUMAN GASTRIC MUCOSA CONTAINING INTRINSIC FACTOR ISOPROTEINS TYPICAL OF MUCOSA. *(E.)* Grasbeck, R.; Marcoullis, G. (Inst. Med. Res., Helsinki, Finland). *Scand. J. Clin. Lab. Invest.* 35(1):13-18, 1975.

A mucosal fraction containing isoproteins of the vitamin B_{12}-intrinsic factor complex with high isoelectric points (pH 5.05-6.52) was isolated from 15 resected stomachs. This fraction (B_{12}-N) was labeled with ^{58}Co-vitamin B_{12}, partially purified by DEAE-cellulose chromatography, and studied by electrofocusing, gel filtration, ultracentrifugation, immunologic procedures, and the Schilling test. The molecular weight of the fraction was about 2000 daltons smaller than gastric juice B_{12}-intrinsic factor. The fraction possessed immunological and biological intrinsic factor activity. When the purified B_{12}-N was mixed with antiserum from a patient with pernicious anemia and subjected to Sephadex G-150 filtration, the radioactivity was totally removed. After administration of 1 µg radioactive vitamin B_{12} bound to the N-component, the fraction of the p.o. dose excreted in the urine of this patient in 2 days was 10.8% compared with 1.1% of unbound vitamin B_{12} (1 µg). Neuraminidase digestion of the gastric juice vitamin B_{12}-intrinsic factor complex produced a biologically active product containing the high pI isoproteins typical of mucosa. Thus, the high pI fraction is deficient in sialic acid and apparently consists of material not yet ready to be secreted. Since sialic acid does not appear to be a biologically active part of the intrinsic factor molecule, it is possible that sialic acid must be removed before intrinsic factor can act on ileal receptors. Although sialic acid-deficient intrinsic factor possessed immunological activity, it is possible that sialic acid-deficient intrinsic factor molecules play a role in initiating intrinsic factor autoantibodies in connection with mucosal inflammatory processes.

5214 ON THE CELLULAR DISTRIBUTION OF SUCRASE AND ENTEROKINASE IN DIFFERENT POPULATIONS OF RAT INTESTINAL EPITHELIAL CELLS ISOLATED BY A VIBRATION METHOD. *(E.)* Schneider, R.; Troesch, V.; Hadorn, B. (Unit Gastro-Enterol., Clin. Univ. Pediatr., Berne, Switzerland). *Biol. Gastroenterol. (Paris)* 8(1):11-20, 1975.

The cellular distribution of protein, enterokinase, and sucrase was studied in isolated rat intestinal epithelial cells. Using a modification of Harrison and Webster's vibration technique, four different populations of cells were obtained from fasted male Wistar DA rats. Fractions 1 and 2 were harvested from the villus, fraction 3 was obtained from the villus-crypt junction, and fraction 4 was obtained from the crypts. The cells were incubated with trypsin or suspended in EDTA, sonicated, treated

with glycodeoxycholate, and dialyzed. The morphology of the small intestinal mucosa before and after removal of the four cell populations was also studied, as was the appearance of the isolated cells. The specific activity of sucrase was significantly and reproducibly different in these fractions, showing the highest specific activity in the cells from the tip of the villus and a steady decrease in activity toward the crypts. There was only a small scatter of results so that the sucrase activity served as a marker for the different cell populations. Enterokinase showed a similar cellular distribution but the scatter of results was much greater and the activities in the various fractions did not always differ significantly. When expressed as percent of total activity, fraction 4 (immature enterocytes) contained more enterokinase than sucrase. When determined in the presence of glycodeoxycholate, there was a 10-fold increase in enterokinase activity. The effect of glycodeoxycholate was due in part to solubilization of the enzyme and in part to an activation of the system enterokinase-trypsinogen. The sucrase activity was not affected by the addition of glycodeoxycholate or preincubation with trypsin. The results suggest that enterokinase is differentiated earlier in the life cycle of the enterocyte than is sucrase.

5215 STRUCTURE OF RAT BILE CANALICULI AS REVEALED BY SCANNING ELECTRON MICROSCOPY. *(E.)* Motta, P.; Fumagalli, G. (Med. Coll., Univ. Rome, Italy). *Anat. Rec.* 182(4):499-514, 1975.

Topographic details of the surface structure of the rat liver bile canaliculus were studied by scanning electron microscopy. Liver tissue from five healthy, young adult rats was perfused with a Tyrode solution and then with 2.5% glutaraldehyde in cacodylate buffer solution. Small pieces of liver were subsequently excised, immersed in the same fixative, and dehydrated prior to microscopic examination. The bile canaliculi appeared as half tubules carved out of the surface of the hepatocytes. In many cases, the bile channel was observed to bifurcate to form two or three long branches running on the same face of the cell. Some of the canaliculi showed a tortuous course with frequent lateral sacculations and/or very short branches; of the lateral branches, some were large and appeared similar to the short lateral branches of the bile canaliculus, while others were smaller and arose obliquely from the subjacent cortical areas of the hepatocyte cytoplasm. The latter aspect was more easily observed in stereo views which showed narrow intracellular projections bordered with a few microvilli opening into the lumen of the acutal intercellular bile canaliculus. Zones measuring up to 5.0 μ in width between the space of Disse and the bile channel were frequently observed. The narrowest of these (0.1 μ) appeared to represent zones where the intercellular clefts arising from the subendothelial space of Disse and its recesses came into close contact with the wall of the bile canaliculus; direct communication between these spaces was not observed. The lateral sacculations observed in some of the bile canaliculi probably correspond to short intracellular branches of the canaliculi.

5216 ENDOCRINE CELLS IN THE HUMAN STOMACH: OPTICAL AND ULTRASTRUCTURAL STUDY IN NORMAL SUBJECTS AND IN SOME PATHOLOGICAL CONDITIONS. *(Fr.)* LeBodic, M. F.; LeBodic, L.; Lefranc, G.; Mussini-Montpellier, J.; Pradal, G.; Tusques, J. (Fac. Med., Nantes, France). *Ann. Anat. Pathol. (Paris)* 19(4);353-370, 1974.

Samples of gastric mucosa obtained from four normal subjects, four patients with gastric ulcers and nine patients with various digestive disorders were used to study human endocrine cells. Based on histochemical and ultrastructural analysis, six distinct types of endocrine cells were found in the human stomach: 1) serotonin cells, exhibiting characteristic yellow fluorescence and containing dense polymorphic granules; 2) enterochromaffin-like cells, essentially located in the fundus; 3) gastrin cells of antropyloric localization characterized by the uneven density of secreting granules; 4) gastrointestinal D cells morphologically related to D cells of islands of Langerhans; 5) infrequent A cells and; 6) "small granule" cells, found more frequently and corresponding to Sasagawa type III cells. Pathological findings in ulcer patients showed a marked decrease in endocrine cells, especially in gastrin cells of the antral mucosa, and the appearance of a large number of very dark endocrine cells which were not found in normal mucosa. Pathological findings in the other patients were too varied to draw valid conclusions, but a large number of dark endocrine cells were also observed in these cases.

5217 ON THE BLOOD SUPPLY OF THE PANCREAS IN CHILDREN. *(Rus.)* Kushch, N. L.; Timchenko, A. D.; Prikhodchenko, V. V. (Dept. Pediatr. Surg., A. M. Gor'kiy Donetsk Med. Inst., USSR). *Klin. Khir.* (3):24-30, 1975.

5218 FRUCTOSE INDUCED GLYCOGENESIS. I. ULTRASTRUCTURAL AND MORPHOMETRIC ANALYSIS OF RAT HEPATOCYTES 7 DAYS AFTER FRUCTOSE OVERLOAD. *(Ger.)* Riede, U. N.; Uhl, H.; Seufer, G.; Robausch, T.; Bohm, N.; Sasse, D. (Pathol. Inst., Univ. Freiburg, Germany). *Beitr. Pathol.* 154(1):63-82, 1975.

5219 THE MORPHOLOGICAL CHANGES IN THE SMALL INTESTINE IN RATS AFTER ITS BLOOD CIRCULATION HAS BEEN TEMPORARILY INTERRUPTED. *(Rus.)* Kulik, V. P.; Gadshieva, Z. M.; Makarova, L. M.; Maksimenkova, A. N. (Dept. Normal Anat., Lumumba Univ., Moscow, USSR) *Arkh. Anat. Gistol. Embriol.* 68(3): 50-56, 1975.

5220 ENDOCRINE CELLS OF THE INTESTINAL MUCOSA. I. ENTEROCHROMAFFIN CELLS. *(Pol.)* Lamprecht, J. (Cell Pathophysiol. Dept., CMKP, Warsaw, Poland). *Patol. Pol.* 25(4):587-594, 1974.

5221 THE EFFECT OF VAGOTOMY ON THE STRUCTURE AND FUNCTION OF THE SMALL INTESTINE. (REVIEW). *(Rus.)* Postolov, P. M.; Fetisov, V. A. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Khirurgiia (Mosk.)* (1):144-150, 1975.

5222 ORGAN CULTURE OF GUINEA PIG AND HUMAN INTESTINAL MUCOSA: ULTRASTRUCTURAL, CYTOCHEMICAL AND BIOCHEMICAL STUDY. *(E.)* Kedinger, M.; Haffen, K.; Hauri, H. P.; Schang, J. C.; Hadorn, B.; Grenier, J. F. (INSERM, Strasbourg, France). *Digestion* 11(5/6):450-451, 1974.

5223 LIGHT MICROSCOPIC MORPHOMETRY AND FINE STRUCTURE OF THE LIVER: A RESPONSE TO PERFLUORINATED LIQUID EMULSIONS USED AS ARTIFICIAL BLOOD. *(E.)* Miller, M. L.; Clark, Jr., L. C.; Wesseler, E. P.; Stanley, L.; Emory, C.; Kaplan, S. (Child. Hosp., Cincinnati, Ohio). *Ala. J. Med. Sci.* 12(1):84-113, 1975.

5224 COMPENSATORY HYPERTROPHY OF THE ILEUM FOLLOWING ESOPHAGOILEOSTOMY. *(E.)* Tilson, M. D.; Sweeney, T.; Wright, H. K. (Yale Univ. Sch. Med., New Haven, Conn.). *Surg. Forum* 24:418-419, 1973.

5225 METHODS OF STUDY OF THE G-CELLS' DISTRIBUTION IN THE NORMAL STOMACH. *(E.)* Potet, F.; Constantin-Voillemot, N.; Lewin, M. (Hosp. Beaujon, Clichy, France). *Digestion* 11(5/6):455, 1974.

5226 DRUG-INDUCED HISTOLOGICAL CHANGES AND ITS CONSEQUENCES ON THE PERMEABILITY OF THE SMALL INTESTINAL MUCOSA. II. *(E.)* Nadai, T.; Kume, M.; Tatematsu, A.; Sezaki, H. (Fac. Pharm., Meijo Univ., Nagoya, Japan). *Chem. Pharm. Bull.* 23(3):543-551, 1975.

5227 MORPHOLOGICAL EFFECTS OF A LARGE SINGLE DOSE OF CYCLOHEXIMIDE ON THE INTESTINAL EPITHELIUM OF THE RAT. *(E.)* Altmann, G. G. (Health Sci. Cent., Univ. West. Ontario, London, Canada). *Am. J. Anat.* 143(2):219-240, 1975.

5228 ULTRASTRUCTURE AND DISTRIBUTION OF ARGYROPHIL AND ARGENTAFFIN CELLS IN RECTUM. *(E.)* Ohisaka, K.; Kimura, K.; Makino, K.; Hokano, M. (Tokyo Med. Coll., Japan). *J. Clin. Microsc.* 7(3-4): 342-343, 1975.

5229 STUDIES OF ETHIONINE PANCREATITIS BY MEANS OF ELECTRON MICROSCOPIC RADIOAUTOGRAPHY. *(E.)* Yoshizawa, S.; Nagata, A.; Homma, T.; Oda, M.; Murata, F.; Nagata, T. (Shinshu Univ. Sch. Med., Matsumoto, Japan). *J. Clin. Electron Microsc.* 7(3-4):349-350, 1974.

5230 CHOLESTEROL CRYSTALS IN THE GALLBLADDER EPITHELIUM AND THEIR IDENTIFICATION BY DIGITONIN. *(E.)* Koga, A.; Nishimura, M. (First Dep. Surg., Kyushu Univ., Japan). *J. Clin. Electron Microsc.* 7(3-4):252, 1974.

5231 SCANNING ELECTRON MICROSCOPIC STUDIES ON BIOPSY SPECIMEN OF DUODENAL MUCOSA-METHODS OF SAMPLE PREPARATION. *(E.)* Ohashi, Y.; Kita, S.; Kobayashi, R.; Kosaka, K. (Okayama Univ. Med. Sch., Japan). *J. Clin. Electron Microsc.* 7(3-4):339-340, 1974.

5232 ELECTRON MICROSCOPIC STUDIES ON THE EXOCRINE CELLS AND THE ENDOCRINE CELLS OF THE RAT STOMACHS AFTER RESTRAINT. *(E.)* Yamaguchi, T.; Tobe, T.; Hikasa, Y.; Sano, M.; Kanato, M. (Second Dep. Surg., Kyoto Univ., Japan). *J. Clin. Electron Microsc.* 7(3-4):337-338, 1974.

5233 INTESTINAL CRYPT SURVIVAL AFTER X-IRRADIATION OF THE RAT SMALL INTESTINE UNDER CONDITIONS OF RADIOPROTECTION. *(E.)* Osborne, J. W.; Bybee, J. W. (Radiat. Res. Lab., Iowa City, Iowa). *Radiat. Res.* 62(3):523, 570, 1975.

5234 MUCOSAL CELL TRANSIT TIME AND COMPENSATORY RESPONSE IN MURINE INTESTINE. *(E.)* Cooper, J. W.; Burholt, D. R.; Hagemann, R. F. (Allegheny Gen. Hosp., Pittsburgh, Pa.). *Radiat. Res.* 62(3): 545, 1975.

5235 A FINE STRUCTURAL STUDY OF ASEXUAL STAGES OF THE MURINE COCCIDIUM Eimeria ferrisi LEVINE AND IVENS 1965. *(E.)* Chobotar, B.; Scholtyseek, E.; Senaud, J.; Ernst, J. V. (Dep. Biol., Andrew's Univ., Berrien Springs, Mich.). *Z. Parasitenk.* 45(4):291-306, 1975.

5236 HISTOCHEMICAL OBSERVATIONS WITH THE LIGHT AND THE ELECTRON MICROSCOPE ON THE MUCOSUBSTANCES OF THE NORMAL MOUSE GALLBLADDER EPITHELIAL CELLS. *(E.)* Wahlin, T.; Bloom, G. D.; Carlsoon, B. (Dep. Pathol. Histol., Univ. Umea, Sweden). *Histochemistry* 42(2):119-131, 1974.

See also, 5245, 5345, 5346, 5378, 5380, 5518, 5567, 5589, 5591, 5592, 5595, 5867, 6031, 6072.

ABSORPTION-EXCRETION-TRANSPORT

5237 THE EFFECT OF VITAMIN A DEFICIENCY ON CELLULAR MEMBRANE PERMEABILITY OF SMALL INTESTINE MUCOSA. *(Ukr.)* Leutskyi, K. M.; Baran, M. M. (Dep. Biochem., Univ. Chernivtsi, USSR). *Ukr. Biokhim. Zh.* 46(1):105-109, 1974.

To elucidate the mechanism of biological functioning of vitamin A, the effect of vitamin A deficiency on the permeability of cellular membranes of small intestine mucosa, and on the intra- and extracellular content of Na, K and water, was studied. Experi-

ments were performed on white rats with initial weight of 50 g, fed on a vitamin A-free diet; the control animals were given daily 250 MU of acetic form of vitamin A. The results showed that A-avitaminosis leads to the increase in membrane permeability of the small intestinal mucosa, to the increase of total and intracellular content of Na ions, to the decrease of total and intracellular content of K ions (thus effecting transmembrane gradient of Na), and to the increase of total and extracellular, but to the decrease of intracellular, content of water.

5238 INTESTINAL ABSORPTION OF CYTIDINE DIPHOSPHATE CHOLINE AND ITS CHANGES IN THE DIGESTIVE TRACT. *(E.)* Yashima, K.; Takamatsu, M.; Okuda, K. (Kurume Univ. Sch. Med., Japan). *J. Nutr. Sci. Vitaminol.* 21(1):49-60, 1975.

The intestinal absorption and metabolism of $^{14}CH_3$ and C_5 pyrimidine ^{3}H-labeled CDP-choline following gastric instillation were studied in fasted, anesthetized Wistar rats. After 3 hr, only 5% of the ^{14}C and 15% of the ^{3}H dose remained in the gut. Hepatic uptake reached a maximum after 2-3 hr. Little to no radioactivity was found in heart, brain, pancreas or urine. Chromatographic analysis of gastric contents 30 min after ^{14}C-CDP-choline instillation showed radioactivity primarily in the CDP-choline fraction whereas intestinal washings showed label primarily in the choline fraction. Label following ^{3}H-CDP-choline instillation was found in gastric CDP-choline and intestinal cytodine. One-half of acid soluble ^{14}C was located in hepatocyte CDP-choline, choline phosphate and choline while most of the ^{3}H was in CMP and cytidine with some in CDP-choline and CDP. These results indicate that CDP-choline is broken down in the intestine to choline and cytidine which are transported and phosphorylated by the intestinal mucosa. The relatively small amounts of $^{14}CO_2$ appearing in expired air (14%) and urine (2%) indicate that only a small amount of choline is completely broken down.

5239 DEPRESSION OF JEJUNAL DIPEPTIDE TRANSPORT BY PYRIDOXINE DEFICIENCY IN THE RAT. *(E.)* Gupta, V. J.; Edwards, K. D. G.; Asatoor, A. M. (Sydney Hosp., Australia). *Aust. J. Exp. Biol. Med. Sci.* 53(1):89-92, 1975.

The intestinal absorption of three dipeptides and a dicarboxylic acid dipeptide differing in mechanisms of membrane transport was studied *in vivo* in 20 fasted, anesthetized vitamin B_6-depleted and B_6-sufficient male Sprague-Dawley rats. Absorption from an isolated 5 cm section of distal duodenum and upper jejunum was significantly slowed by 50-60% in B_6-depleted animals compared to B_6-sufficient rats. These results confirm previous ones in which decreased intestinal absorption of L-alanyl-L-phenylalamine glycylglycine and L-leucyl-L-tyrosine was found in B_6-depleted rats. Analysis of variance regression lines for dipeptide and amino acid transport in the present system provided support for the concept that dipeptide transport, like amino acid transport, is non-selectively depressed by B_6 deficiency. The results further suggest that both dipeptide and amino acid transport are mediated by a common step which is B_6-dependent.

5240 DIPEPTIDE TRANSPORT IN ISOLATED INTESTINAL BRUSH BORDER MEMBRANE. *(E.)* Sigrist-Nelson, K. (Lab. Biochem., Fed. Inst. Technol., Zurich, Switzerland). *Biochim. Biophys. Acta* 394(2):220-226, 1975.

The transport of glycyl-L-leucine into isolated brush border membrane vesicles was studied in Sprague-Dawley rats. Highly purified brush border membranes from isolated epithelial cells of the rat small intestine were incubated in media containing mannitol, buffer, $MgSO_4$, and (1-^{14}C)glycyl-L-leucine, glycyl-L-(U-^{14}C)leucine, (2-^{3}H)glycine, and/or L-(4,5-$^{3}H_2$)-leucine. At equilibrium, the uptake values for glycyl-L-leucine (labeled in the glycine and leucine moiety), glycine, and L-leucine were the same; the uptake of both labeled species of dipeptide being similar, while the time courses for glycine and L-leucine were markedly different. Na^+ stimulated L-leucine transport, had a lesser effect on transport of the dipeptide and did not affect glycine uptake. When the membranes were incubated with the radioactive dipeptide, 80% of the initial radioactive content was dipeptide and 20% was glycine; after 5 min, however, 95% of the radioactivity was in the form of glycine and only 5% was in the form of dipeptide. The uptake of glycyl-L-leucine was inhibited by other dipeptides, particularly leucyl and alanyl peptides, but was unaffected by glycine and L-leucine. Glycyl-L-leucine stimulated the uptake of glycine and L-leucine, the stimulatory effect increasing with time. The data support the concept that glycyl-L-leucine is primarily transported intact across the brush border membrane by a specific mechanism differing from that involved in amino acid transport.

5241 A COMPARATIVE STUDY ON THE EFFECTS OF DIFFERENT BILE SALTS ON MUCOSAL ATPase AND TRANSPORT IN THE RAT JEJUNUM IN VIVO. *(E.)* Guiraldes, E.; Lamabadusuriya, S. P.; Oyesiku, J. E. J.; Whitfield, A. E.; Harries, J. T. (Inst. Child Health, London, England). *Biochim. Biophys. Acta* 389(3):495-505, 1975.

Closed-loop and perfusion techniques were used to study the effects of deoxycholate, taurocholate and cholate on mucosal ATPase and transport activity in the rat (Wistar strain) jejunum. In the closed-loop experiments, a 25-min absorptive period was used, while in the perfusion studies the infusate flow rate was 0.2 ml/min. In the closed loops, 5 mM deoxycholate selectively inactivated ($Na^+ + K^+$)-ATPase, and a net secretion of Na^+ induced by 2.5 mM deoxycholate was due to reduced lumen to plasma flux of the ion. Deoxycholate (2.5 mM) also markedly inhibited 3-O-methylglucose transport. The luminal disappearance rates of deoxycholate (60.5%/g) greatly exceeded those of taurocholate (4.3%/g). In the perfusion studies, 1 mM of deoxycholate induced a net secretion of water, Na^+ and Cl^- and inhibited active glucose trans-

port; "total" ATPase, ($Na^+ + K^+$)-ATPase and Mg^{2+}-ATPase were also inhibited. At higher concentrations (5 mM), deoxycholate stimulated Mg^{2+}-ATPase activity. Taurocholate and cholate had no effect on transport or ($Na^+ + K^+$)-ATPase, and the activities of mucosal lactase, sucrase, and maltase were not affected by 1 mM deoxycholate, taurocholate, or cholate. The mean disappearance rates of deoxy(^{14}C)cholate, cholate, and taurocholate from the intestinal lumen were 50.0%, 30.0%, and 17.7%/g/min. The results suggest that deoxycholate inhibits sodium-coupled glucose transport *via* inhibition of ($Na^+ + K^+$)-ATPase at the lateral and basal membranes of the epithelial cells, rather than from an effect at the brush-border membrane level. ($Na^+ + K^+$)-ATPase may play a fundamental role in the physiology of intestinal transport, and inhibitory substances may be important in the pathophysiology of certain types of diarrhea.

5242 A GLUCOSE-STIMULATED NONELECTROGENIC SODIUM PUMP IN ILEAL REMNANTS AFTER JEJUNECTOMY. *(E.)* Tilson, M. D.; Clifford, B. (Yale Univ. Sch. Med., New Haven, Conn.). *J. Surg. Res.* 13(4):405-408, 1975.

To determine whether "jejunization" of ileal transport processes may occur after proximal resection, the glucose dependence of sodium transport and the transmural potential differences (PDs) were studied in ileal remnants after jejunectomy in adult Sprague-Dawley rats. Fourteen to 21 days after jejunectomy or sham jejunectomy, an isolated distal ileal segment was perfused with test solutions containing mannitol or glucose. Effluent samples were collected at 30-min intervals and the PD was measured every 5 min. The PD of normal ileum increased from 6.44 with mannitol to 7.70 after perfusion with glucose solution. The PD of ileum from the jejunectomized rats decreased from 4.86 with mannitol to 4.6 with glucose, the mean PD of glucose-stimulated normal ileum being significantly greater. The net sodium flux increased nonsignificantly after glucose stimulation in normal animals, the glucose-stimulated increase being significant in the jejunectomized animals. Net Na absorption was a linear function of water flux in both the normal and jejunectomized animals. These observations are consistent with the hypothesis that the transport process of the adapted ileal remnant may begin to imitate those characteristics of the resected segment, a process which has been termed "jejunization."

5243 THE ACTIVATION OF INTESTINAL BRUSH BORDER SUCRASE BY ALKALI METAL IONS: AN ALLOSTERIC MECHANISM SIMILAR TO THAT FOR THE NA^+-ACTIVATION OF NONELECTROLYTE TRANSPORT SYSTEMS IN INTESTINE. *(E.)* Mahmood, A.; Alvarado, F. (Sch. Med., Univ. Puerto Rico, San Juan). *Arch. Biochem. Biophys.* 168(2):585-593, 1975.

A model for the activation of intestinal brush border sucrase by alkali-metal ions was applied to new data on the mechanism of the Na^+-activation of brush border sucrase from the guinea pig intestine. The model describes the activation by alkali-metal ions by an allosteric, noncompulsory mechanism involving two distinct sites: one for sucrase and another for the metal activator. In the present experiment, sucrase activity was determined with glucose oxidase-peroxidase in guinea pig brush borders incubated in 4 mM Li-succinate containing 20 mM sucrose and Na^+, K^+, and/or Li^+. Both Na^+ and K^+ activated the guinea pig sucrase, but K^+ had 10 times more affinity for the metal activator site; Li^+ was inert. Values for the dissociation constants for the interaction of sucrase with sucrose, Na^+, or K^+ were calculated for these data and for previously recorded data for the rat and hamster. Qualitatively, the activation of sucrase by alkali metal ions was similar to that described for the Na^+-activation of amino acid and sugar transport in the intestine. However, the Na^+-binding site in the two systems was apparently quite different. In the guinea pig, the Na^+-dissociation constant was on the order of 10^{-3} M for sucrase, about two orders of magnitude smaller than that for transport. Also, K^+ was a strong activator of guinea pig sucrase, but was an inhibitor of intestinal sugar transport. It is concluded that the brush border sucrase and sugar carrier in the intestinal sugar transport system do not share a common Na^+-binding site.

5244 ABSORPTION OF TWO PROLINE CONTAINING PEPTIDES BY RAT SMALL INTESTINE IN VIVO. *(E.)* Lane, A. E.; Silk, D. B. A.; Clark, M. L. (St. Bartholomew's Hosp., London, England). *J. Physiol.* 248(1):143-149, 1975.

The *in vivo* intestinal absorption of glycyl-L-proline and L-prolyl-glycine, two dipeptides which are poorly hydrolyzed *in vitro* by brush border peptide hydrolases, was investigated in adult male Wistar rats. A steady-state perfusion technique was used and jejunal absorption was compared with ileal absorption using the rates of absorption of constituent amino acids from dipeptide and equivalent equimolar amino acid solutions. Glycine and proline were absorbed at the same or faster rates during perfusion of 10 mM glycyl-L-proline than during the perfusion of its equivalent equimolar amino acid solution. Conversely, the two constituent amino acids were absorbed at slower rates during the perfusion of 10 mM L-prolyl-glycine than during perfusion of its equivalent amino acid solution. As expected, very low concentrations of free amino acids were detected during the jejunal dipeptide perfusions, while higher concentrations were detected during the ileal perfusions. The addition of 40 mM L-prolyl-glycine to the 10 mM glycyl-L-proline solution did not affect the rates of disappearance of glycyl-L-proline. The addition of 40 mM of glycyl-L-proline to 10 mM L-prolyl-glycine, however, had a significant inhibitory effect on the disappearance of L-prolyl-glycine. It is concluded that whereas glycyl-L-proline and L-prolyl-glycine are absorbed predominantly intact in the jejunum, ileal absorption probably involves both direct peptide uptake and absorption of liberated amino acids by specific amino acid transport systems.

5245 MORPHOLOGICAL AND FUNCTIONAL EFFECTS OF BILE SALTS ON RAT COLON. *(E.)* Saunders, D. R.; Hedges, J. R.; Sillery, J.; Esther, L.; Matsumura, K.; Rubin, C. E. (Dep. Med., Univ. Washington, Seattle). *Gastroenterology* 68(5):1236-1245, 1975.

The effects of conjugated and unconjugated bile salts on the morphology of and net water transport across the rat colon were measured *in vivo*. Colonic segments, consisting of the large intestine distal to the cecum, were infused in an isoperistaltic direction in anesthetized male Sprague-Dawley rats. The test infusates consisted of buffered saline, delipidated rat bile (7-9 mM bile salts), 1-3 mM deoxycholate, or 2.5-10 or more mM taurocholate. Epithelial alteration in the colonic absorptive cells was equivocal and water absorption was uninhibited during infusions of delipidated rat bile, 1 mM deoxycholate, or of concentrations of taurocholate less than 10 mM. In contrast, 3 mM deoxycholate and taurocholate in concentrations of 10 mM or greater caused severe alterations in the colonic epithelium as seen under the light microscope, and inhibited water absorption. Under the electron microscope, the absorptive cells covering the colonic mucosal surface of segments infused with higher concentrations of deoxycholate or taurocholate were reduced in height and many showed structural signs of cell injury. Dilation of intercellular spaces was an inconsistent finding which correlated poorly with the rate of net water transport. The results suggest that in some patients with ileal disease, abnormally high bile salt concentrations in the fecal water would be expected to inhibit water absorption and perhaps to alter the structure of colonic absorptive cells.

5246 FATTY ACID INHIBITION OF WATER ABSORPTION AND ENERGY PRODUCTION IN THE HAMSTER JEJUNUM. *(E.)* Gaginella, T.; Bass, P.; Olsen, W.; Shug, A. (Sch. Pharm., Univ. Wisconsin, Madison). *FEBS Lett.* 53(3):347-350, 1975.

To determine the mechanism involved in the fatty acid inhibition of intestinal water and sodium absorption, the effects of hydroxylated and nonhydroxylated octadecenoates on intestinal water transport and adenine nucleotide translocase (ANT) activity were studied in nonfasted male Golden Syrian hamsters. Water transport was assessed using everted gut sacs prepared from 12 cm segments of jejunum distal to the ligament of Treitz. The sacs were incubated with continuously gassed mucosal and serosal buffer solutions containing the fatty acids to be tested. ANT activity was measured directly in the homogenates of mucosal scrapings of sacs incubated without polyethylene glycol. Purified mitochondria were also prepared for respiratory studies. Ricinoleate, ricinelaidate, and oleate inhibited ANT activity and water transport, the latter being a stronger inhibitor of ANT and the two former fatty acids being stronger inhibitors of water transport. Elaidate inhibited neither ANT nor water transport. The ANT-specific inhibitor atractylate inhibited ANT activity by about 90%, but inhibited water transport by only 40%. Oleoyl CoA inhibited respiration in the purified mitochondria, inhibition of the coupled state being reversed by the addition of the uncoupler CICCP; similar inhibitory effects were observed with atractylate. These results indicate that in the intestine, as in other organs, ANT, by controlling the mitochondrial ATP/(ADP + P_i) ratio can influence the regulation of energy-linked cellular metabolism. This in part accounts for the effects of fatty acids on intestinal water absorption.

5247 THIAMINE TRANSPORT ACROSS THE RAT INTESTINE. I. NORMAL CHARACTERISTICS. *(E.)* Hoyumpa, A. M., Jr.; Middleton, H. M.; Wilson, F. A.; Schenker, S. (Vanderbilt Univ. Med. Sch., Nashville, Tenn.). *Gastroenterology* 68(5):1218-1227, 1975.

The characteristics of normal thiamine transport across the intestine were studied in female Sprague-Dawley rats using intact intestinal loops and everted jejunal segments. *In vivo* studies with (^{35}S)thiamine hydrochloride revealed, in all segments of the small intestine, saturation kinetics for low thiamine concentrations (0.06-1.5 μM), with a linear relationship between higher concentrations (2-560 μM) and absorption. Moreover, *in vitro* studies of net transmural flux using everted jejunal sacs demonstrated movement of (^{14}C)thiamine hydrochloride against a concentration gradient only when low thiamine concentrations were used, so that the serosal to mucosal ratio became significantly greater than the initial value of 1. Pyrithiamine (2 μM), dinitrophenol (200 μM), norethylmaleimide (100 μM), and ouabain (100 μM) reduced the net transmural flux of 0.2 μM thiamine. When unidirectional flux across the jejunum was measured, saturation kinetics were again demonstrated for low thiamine concentrations. This phenomenon, however, was abolished by the addition of pyrithiamine, which exerted competitive inhibition on thiamine absorption. Anoxia and sodium lack reduced the intestinal uptake of 0.5 μM thiamine to 58% and 74%, resp., or normal, but did not affect the uptake of 50 μM thiamine. Lowering the temperature led to a fall in thiamine uptake, the reduction being more marked with low thiamine concentrations (Q_{10}, 1.648) than with high concentrations (Q_{10}, 1.127). Stirring the water layer reduced the K_m to 59% of the unstirred value, while the V_{max} and permeability coefficient remained unchanged. Finally, movement of low concentrations of thiamine against an electrical gradient was observed under conditions of electrical short circuiting and zero potential difference; no such effect was seen with high concentrations. These studies suggest that there exists in the rat a dual system of intestinal thiamine transport: at low concentrations, thiamine is absorbed by an active process; and at high concentrations, transport across the intestine is largely a passive movement.

5248 MUCOSAL UPTAKE IN VITRO OF CHOLESTEROL FROM BILE SALT AND SURFACTANT SOLUTIONS. *(E.)* Feldman, E. B.; Cheng, C. Y. (State Univ. New York Downstate Med. Cent., Brooklyn). *Am. J. Clin. Nutr.* 28(7):692-698, 1975.

Cholesterol influx rates were analyzed across rat jejunum from various mixed micellar solutions of conjugated bile salts. Rat jejunal mucosal sheets were exposed for 1-2 min to 10 mM micellar solutions of taurodeoxycholate, dodecylsulfate, Tween 80, or cetyltrimethylammonium bromide containing cholesterol, 0.25 mM. Monoolein and/or oleic acid were variably added. Cholesterol influx was insignificant in all solubilizers without additives. With taurodeoxycholate, addition of monoolein progressively enhanced influx from 13 nM/cm^2/hr to 29 nM/cm^2/hr (0.67-5.4 mM monoglyceride). Oleic acid, 3 mM, was as effective as 5.4 mM monoolein. Oleic acid, 3 mM, added to monoolein, 3 mM, maximized influx (42 nM/cm^2/hr). With cetyltrimethylammonium bromide, monoolein, 0.67 mM, enhanced influx to 13 nM/cm^2/hr, but further additions of monoolein with or without oleic acid had no added effect. Additives had no significant effect on the influx from the other anionic or nonionic surfactants. The results suggest that factors other than cholesterol, monoolein or oleic acid partition, and micellar size may be determinants of sterol influx, such as specific interactions of bile salt and fatty acid or monoglyceride with the plasma membrane.

5249 EFFECT OF PERFUSION RATE ON ABSORPTION, SURFACE AREA, UNSTIRRED WATER LAYER THICKNESS, PERMEABILITY, AND INTRALUMINAL PRESSURE IN THE RAT ILEUM IN VIVO. *(E.)* Lewis, L. D.; Fordtran, J. S. (Univ. Texas Health Science Center, Dallas, Tex.). *Gastroenterology* 68(6):1509-1516, 1975.

The effect of perfusion rate on the absorption of glucose, tritiated water, urea and mannitol was studied in the rat ileum *in vivo*. Groups of eight female Sprague-Dawley rats were perfused with a test solution (each containing 5 g/l of polyethylene glycol as a marker). The first series of experiments utilized a partially obstructed intestinal segment. Glucose absorption from a 72 ml glucose solution and tritiated water absorption increased equally at about 7% as perfusion rate was increased from 10 to 100 ml/min. Glucose absorption from a 4 mM solution was increased by 70% as perfusion rate was increased from 10 to 100 ml/min. Mean intraluminal pressure was 2.9 mm Hg at a perfusion rate of 1 ml/min and increased only slightly when the rate was 10 ml/min. With perfusion rates of 100 and 200 ml/min, pressure rose to 7.5 and 17.8 mm Hg, resp. Because mannitol absorption was 0 at all perfusion rates, none of the enhanced glucose absorption rates was attributed to enhanced mucosal permeability, even though intraluminal pressure was increased at higher perfusion rates. Urea absorption was influenced by surface area and by permeability changes, but not by the thickness of the unstirred water layer. The effect of unstirred water layer thickness on the inhibitory effect of sodium replacement by magnesium on glucose absorption from a 4 mM glucose solution was also studied. Inhibition by sodium removal was equal at 1, 10, 100 and 200 ml/min. Experiments in an unobstructed intestinal segment revealed that the major effect of enhanced perfusion rate was to increase mucosal surface area. These data emphasize the need for a surface area marker in studies done to evaluate the effect of perfusion rate on intestinal absorption *in vivo*, and tritiated water absorption seems preferable to maximal glucose transport rates.

5250 IS PHLORIZIN OR ITS AGLYCON THE INHIBITOR OF INTESTINAL GLUCOSE TRANSPORT? A STUDY IN NORMAL AND LACTASE DEFICIENT MAN. *(E.)* Blum, A. L.; Haemmerli, U. P.; Lorenz-Meyer, H. (Med. Clin., Stadtspital Triemli, Zurich, Switzerland). *Eur. J. Clin. Invest.* 5(3):285-288, 1975.

The inhibition of intestinal glucose absorption by phlorizin was studied in ten normal and six lactase-deficient human subjects. Small bowel perfusion studies were performed with a 4-lumen tube, which was introduced into the intestine after an overnight fast; the proximal balloon of the apparatus was placed at the beginning of the third part of the duodenum. For the first 80 min, an isotonic solution containing 167 mM of glucose, 77 mM of NaCl, and 10 g/l of polyethylene glycol was perfused at a rate of 10 ml/min; in some subjects, 2 mM of phlorizin were added to this solution during the following 80-min period. Intestinal aspirates were collected throughout the experiment and glucose content was measured using the hexokinase method. During the control period, glucose absorption was 8.57 g/hr in the controls and 9.99 g/hr in the subjects with lactase deficiency. Steady state was not reached in the perfused segments during this period. Following phlorizin administration, there was a rapid reduction in glucose absorption, which reached, at the end of this period, 31.5% of the mean absorption rate before phlorizin in the controls and 33.0% in the lactase-deficient subjects. In normal subjects receiving no phlorizin, the absorption rate remained constant during the second period. The results suggest that phlorizin directly inhibits glucose transport in man and that phlorizin hydrolase is not necessary for this effect.

5251 SUGAR ABSORPTION BY FOETAL AND NEONATAL RAT INTESTINE IN VITRO. *(E.)* Bamford, D. R.; Ingham, P. A. (Dep. Zool., Queen's Univ., Belfast, Ireland). *J. Physiol. (Lond.)* 248(2):335-348, 1975.

The development of jejunal sugar absorption by fetal and neonatal rat intestine was studied using an *in vitro* accumulation technique. Jejunal segments from fetal and neonatal rats (aged 5 to 26 days) were incubated for 30 min in 5.0 mM of D-galactose. The highest level of uptake was just before birth and throughout the first week post partum. The integrity of the absorbed sugar was checked by chromatography; no appreciable metabolism of galactose took place during the incubations. When rings of fetal or neonatal intestine were incubated in 2,4-dinitrophenol, significant reductions (101.92 to 66.7 μM/g) in galactose uptake were observed. Before "cut-off" (the time at which the absorption of intact protein ceased), the ileum was significantly more effective in galactose absorption than the jejunum or duodenum. Around "cut-off" the jejunum became the principal site of absorption. The adult pattern of functional localization along the intestine was not fully established until after the third week post partum. The results suggest that variations with respect to

age in the distribution or density of the sugar carriers along the intestine, rather than changes in the nature of the carriers themselves, account for the variation in sugar absorption.

5252 THE EFFECT OF RAISING THE CONCENTRATION OF POTASSIUM IN THE BLOOD OR INTESTINAL LUMEN ON THE TRANSPORT OF POTASSIUM IN THE DOG INTESTINE. *(E.)* Hardy-Smith, A.; Shields, R. (Dep. Surg., Univ. Liverpool, England). *Br. J. Surg.* 62(6):455-461, 1975.

The rates of transport of potassium, sodium, and water across the intestinal mucosa of Thiry-Vella fistulas of colon and ileum were studied in four dogs over a period of several months under three conditions: control, following i.v. infusion of potassium (15 mEq) over 30 min, and during variations in the intraluminal concentration of potassium (0.8-16.0 mEq/l). Following i.v. infusion of potassium, the rates of potassium secretion into the colon were usually increased; the rates of sodium and water absorption were either unchanged or decreased. The unidirectional rate of movement of potassium into the lumen increased as the concentration difference across the mucosa increased; the movement of potassium in the opposite direction remained relatively constant. The unidirectional movement of sodium and water into the lumen of the colon also increased but to a lesser extent. The effect on potassium transport in the ileum was not significant. With increase in the intraluminal concentration of potassium, there was a significant decrease in the rate of net potassium secretion in both colon and ileum. The rates of net absorption of sodium and water were not significantly altered. Unidirectional potassium movement into the body increased in all the ileal fistulas. The results suggest that whenever the concentration of potassium in the plasma rises, potassium secretion into the gastrointestinal lumen is increased.

5253 INTESTINAL AMINO ACID ABSORPTION IN VITAMIN D DEPENDENT RICKETS. *(Ger.)* Harmeyer, J.; Martens, H.; Vogelgesang, G. (Physiologisches Inst. der Tierarztlichen Hochschule, D-3000 Hannover, Bischefshofer Damm 15, Germany). *Z. Kinderhoilkd.* 119(4):245-251, 1975.

5254 DISULFIRAM AND GASTROINTESTINAL MOTILITY: EFFECT OF ETHANOL ABSORPTION IN THE RAT. *(Fr.)* Lamboeuf, Y.; De Saint Blanquat, G.; Derache, R. (Inst. Physiol., Toulouse, France). *Arch. Int. Pharmacodyn. Ther.* 213(1):163-169, 1975.

5255 EFFECT OF A DEGLYCYRRHIZINISED LIQUORICE COMPOUND ON THE GASTRIC MUCOSAL BARRIER OF THE DOG. *(E.)* Morris, T. J.; Calcraft, B. J.; Rhodes, J.; Hole, D.; Morton, M. S. (Univ. Hosp. Wales, Cardiff). *Digestion* 11(5/6):355-363, 1974.

5256 EFFECTS OF CHOLERA TOXIN ON TRANSCELLULAR Na TRANSPORT OF RABBIT ILEUM. *(E.)* Desjeux, J. F.; Curran, P. F. (Hosp. Herold, INSERM U-83, Paris, France). *Digestion* 11(5/6):443, 1974.

5257 EFFECT OF GLUCOSE ABSORPTION ON POTASSIUM MOVEMENTS IN THE HUMAN JEJUNUM. *(E.)* Bernier, J. J.; Modigliani, R.; Matuchansky, C.; Rambaud, J. C. (INSERM U-54, Hosp. St. Lazare, Paris, France). *Digestion* 11(5/6):439, 1974.

5258 EFFECTS OF CARBENOXOLONE SODIUM AND OF ASPIRIN ON GASTRIC MUCOSAL POTENTIAL DIFFERENCE IN NORMAL SUBJECTS. *(E.)* Bennett, P. N.; Frigo, G. M.; Weerasinghe, W. M. T.; Lennard-Jones, J. E. (Univ. Coll. Hosp., London, England). *Scand. J. Gastroenterol.* 10:161-164, 1975.

5259 DUODENAL HORMONES AND INTESTINAL POTENTIAL DIFFERENCE. *(E.)* Wingate, D. L.; Cooper, W. (London Hosp. Med. Coll., England). *Digestion* 11(5/6):462, 1974.

5260 EFFECT OF AN ULCER ON THE GASTRIC MUCOSAL BARRIER IN DOGS. *(E.)* Cooke, A. R.; Kienzle, M. G.; Tubbesing, T. J. (Veterans Hosp., Iowa City, Iowa). *Digestion* 11(5/6):347-354, 1974.

5261 EFFECT OF FREE AND BOUND GOSSYPOL ON THE ABSORPTION OF L-(^{14}C)LYSINE, L-(^{14}C)-METHIONINE, AND L-(^{14}C)VALINE FROM THE RAT SMALL INTESTINE. *(E.)* Jones, L. A.; Smith, F. H. (Dep. Animal Sci., North Carolina State Univ., Raleigh). *J. Agr. Food Chem.* 23(4):647-653, 1975.

5262 MECHANISM OF THE INTESTINAL ABSORPTION OF DRUGS FROM OIL IN WATER EMULSIONS. V. ENHANCED ABSORPTION OF METHYL ORANGE ADSORBED AT OIL/WATER INTERFACE IN EMULSIONS. *(E.)* Ogata, H.; Kakemi, K.; Furuya, A.; Fujii, M.; Muranishi, S.; Sezaki, H. (Fac. Pharm. Sci., Kyoto Univ., Japan). *Chem. Pharm. Bull.* 23(4):716-724, 1975.

5263 CALCIUM UPTAKE BY ISOLATED SMALL INTESTINAL BRUSH BORDER MEMBRANES FOLLOWING DIETARY CALCIUM RESTRICTION. *(E.)* Krawitt, E. L.; Katagiri, C. A. (Dep. Med., Univ. Vermont, Burlington, Vt.). *Gastroenterology* 68(4):930, 1975.

5264 CHANGES IN INTESTINAL TRANSPORT FOLLOWING JEJUNOILEAL BYPASS SURGERY. *(E.)* Kozloff, I.; Jackson, M. J.; Joseph, W. L. (George Washington Univ. Med. Cent., Washington, D.C.). *Gastroenterology* 68(4):929, 1975.

5265 THE EFFECTS OF ACUTE ALTERATIONS IN HEMODYNAMICS, OXYGEN AVAILABILITY AND ACID BASE BALANCE ON THE PERMEABILITY OF THE GASTRIC MUCOSA. *(E.)* Ritchie, Jr., W. P. (Dep. Surg., Univ. Virginia, Charlottesville). *U.S. Dept. Comm. Nat. Tech. Infor. Serv.* 75(12):40, 1975.

5266 PROTEIN ABSORPTION BY NEONATAL PUPS THROUGH TWO WEEKS OF NEONATAL LIFE. *(E.)* Staley, T. E. (Coll. Vet. Med., Oklahoma State Univ., Stillwater). *Am. J. Dig. Dis.* 20(6):596-597, 1975.

See also, 5269, 5568, 5580.

MOTILITY

5267 SELECTIVE ANTAGONISM OF THE INTESTINAL STIMULATORY EFFECTS OF MORPHINE BY ISOPROTERENOL, PROSTAGLANDIN E_1 AND THEOPHYLLINE. *(E.)* Grubb, M. N.; Burks, T. F. (Univ. Texas Med. Sch., Houston). *J. Pharmacol. Exp. Ther.* 193(3):884-891, 1975.

Studies were undertaken to determine whether treatment with isoproterenol, prostaglandin E_1 and theophylline would inhibit intestinal responses to morphine without interfering with motor responses to acetylcholine. Contractions (induced by graded, intraarterial bolus doses of morphine and acetylcholine) were measured in isolated segments of adult mongrel dog intestine perfused *via* the vasculature with Kreb's solution. Addition of the α- and β-adrenergic receptor agonist, norepinephrine (0.2 µg/ml), to the perfusion solution slightly decreased the magnitude of contractor responses to acetylcholine; responses to morphine were decreased considerably. The pure α-adrenergic receptor agonist, methoxamine (50 µg/ml), and the pure β-adrenergic receptor agonist, isoproterenol (5 µg/ml), produced similar effects. Perfusion of intestinal segments with Kreb's solution containing prostaglandin E_1 (1 µg/ml) or theophylline (180 µg/ml) resulted in marked decreases in responses to morphine but had no significant effects on responses to acetylcholine. Patterns of inhibition similar to those seen with morphine have been previously observed with 5-hydroxytryptamine, which may mediate the intestinal stimulatory effects of morphine. The mild inhibitory effects of methoxamine and isoproterenol on responses to acetylcholine are attributed to actions on neural and possibly non-neural adrenergic receptors. Since isoproterenol, prostaglandin E_1 and theophylline have in common the ability to elevate or maintain intracellular levels of cyclic AMP (cAMP), it is suggested that the intestinal stimulatory effects of morphine may result from 5-hydroxytryptamine-mediated interference with smooth muscle inhibitory actions of cAMP.

5268 INTERACTION BETWEEN ALUMINUM AND CALCIUM IONS IN POTASSIUM CHLORIDE DEPOLARIZED GASTROINTESTINAL SMOOTH MUSCLE. *(E.)* Hava, M.; Hurwitz, A. (Univ. Kansas Med. Cent., Kansas City). *Arch. Int. Pharmacodyn. Ther.* 214(2):213-223, 1975.

The effects of aluminum chloride and lanthanum chloride on gastrointestinal smooth muscle (under varying conditions of depolarization) were described, in order to define the interaction of these trivalent cations with calcium. Isolated colon (2-3 cm portions) from male Sprague-Dawley rats, and segments from guinea pig ileum were used. Aluminum chloride (5×10^{-4} M) abolished the inhibitory effects of papaverine chloride (1.5×10^{-5} M) on contractions of ileum and colon evoked by calcium in Tyrode's solution containing a high concentration of KCl. The inhibitory effects of cocaine hydrochloride (10^{-3} M) and $MgSO_4$ (2×10^{-2} M) were not antagonized. In isolated colon (depolarized by partial exchange of NaCl by 40 mM KCl), $AlCl_3$ decreased the phasic contractions and slowed the onset of tonic contraction during increase of the $CaCl_2$ concentration from 1.8×10^{-3} M to 7.2×10^{-3} M. The increase in spontaneous activity was not blocked by atropine sulfate (10^{-7}–10^{-4} M), but was partially antagonized by 10^{-3} M caffeine and was completely blocked by papaverine chloride (5×10^{-4} M). More than one site of action is proposed for the effects of aluminum on gastrointestinal smooth muscle.

5269 INHIBITION OF SMALL INTESTINAL MUCOSAL AND SMOOTH MUSCLE CELL FUNCTION BY RICINOLEIC ACID AND OTHER SURFACTANTS. *(E.)* Gaginella, T. S.; Stewart, J. J.; Gullikson, G. W.; Olsen, W. A.; Bass, P. (Cent. Health Sci., Univ. Wisconsin, Madison). *Life Sci.* 16(10):1595-1606, 1975.

The effects of ricinoleic acid on the inhibition of net water and on electrolyte transport and intestinal smooth muscle contractile response *in vitro* were compared with the effects produced by two structurally different, anionic surfactants, a non-ionic surfactant and two bile salts. Net water and electrolyte transport from everted hamster (Golden Syrian) gut sacs and contractile activity of electrically-stimulated guinea pig ileum were assessed. Sodium ricinoleate, dioctyl sodium sulfosuccinate, sodium dodecyl (lauryl) sulfate, polysorbate 80, sodium deoxycholate and chenodeoxycholate were found to produce depression of *in vitro* mucosal and smooth muscle cell function. All compounds were effective depressants of both systems at concentrations which were likely to be below their respective critical micellar concentrations. It is suggested that ricinoleic acid may produce its cathartic effect due to its amphipathic nature, possibly by hydrophobic interaction with membrane lipoproteins. Ricinoleic acid and the other surfactants may be acting through a common mechanism.

5270 EFFECTS OF BOMBESIN AND BOMBESIN-LIKE PEPTIDES ON GASTROINTESTINAL MYOELECTRIC ACTIVITY. *(E.)* Caprill, R.; Melchiorri, P.; Improta, G.; Vernia, P.; Frieri, G. (Second Med. Clin., Rome, Italy). *Gastroenterology* 68(5):1228-1235, 1975.

The effects of bombesin and bombesin-like peptides on the electrical activity in the gastrointestinal tract were studied in conscious mongrel dogs. Electrodes were chronically implanted in the intestinal muscle layer at different levels between the stomach and rectum. Bombesin (5, 10, 15, 30 ng/kg/min), COOH-terminal heptapeptide and COOH-terminal octapeptide (20 and 80 ng/kg/min), COOH-terminal nonapeptide (10, 15, 30 ng/kg/min), and litorin (a new natural peptide isolated from the skin of *Litoria aurea*, 10, 20, 30, 40 ng/kg/min were infused for 20 min into the saphenous vein. The effects of the drugs on frequency, amplitude, and rhythm of pacesetter potentials (PP) and incidence of spikes were evaluated. Bombesin significantly increased the frequency of PP in the antrum, duodenum, jejunum, and ileum. In the duodenum and jejunum, the increase in PP frequency showed a linear correlation with the reduction in PP amplitude, the propagation velocity of PP also being clearly reduced. Spikes were not affected in the antrum and ileum, whereas they were abolished in the duodenum and jejunum. In the duodenum, the increase in PP frequency and the slowing down of the propagation velocity were followed by a loss of PP phase lock and the appearance of a characteristic electrical pattern consisting of an irregular sequence of small and slow potentials ("electric disorganization"). The mechanical counterpart was the disappearance of intraluminal pressure activity. In the colon, the effect of bombesin on electrical activity was inconsistent. Neither the COOH-terminal heptapeptide nor the octapeptide of bombesin showed a significant effect on myoelectric activity, whereas the effects of COOH-terminal-nonapeptide and litorin were similar to those of bombesin. Thus, the characteristic electrical changes of PP produced by bombesin appear to be related to the sequence of the 9 amino acids in the COOH-terminal residue of the bombesin molecule.

5271 INHIBITION OF GASTRIC EMPTYING IS A PHYSIOLOGICAL ACTION OF CHOLECYSTOKININ. *(E.)* Debas, H. T.; Farooq, O.; Grossman, M. L. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Gastroenterology* 68(5):1211-1217, 1975.

The relative effects of cholecystokinin (CCK) and gastrin on gastric emptying in the dog were compared to determine whether CCK plays a physiological role in the inhibition of gastric emptying. Dogs were prepared with gastric fistulas, gastric fistulas and chronic pancreatic fistulas, or gastric fistulas and chronic bile fistulas. To stimulate physiological conditions, CCK, OP-CCK (synthetic C-terminal octapeptide CCK), and 20% CCK were administered by continuous i.v. infusion in doses known to be submaximal for pancreatic protein secretion and gallbladder contraction (0.75-3.0 U/kg/hr for CCK, 1.5-6.0 U/kg/hr for 20% CCK, and 63-250 ng/kg/hr for OP-CCK); in addition, L-tryptophan (10-40 mM/liter) was administered to determine the action of endogenous CCK. The effect of endogenously released CCK was studied by measuring the rate of emptying of NaCl solutions in which different concentrations of tryptophan replaced equiosmolar amounts of NaCl, the rate of emptying of the NaCl solution also being measured after exogenous administration of the test drugs. The D_{50}'s of 20% CCK (3 U/kg/hr) and of OP-CCK (125 ng/kg/hr) for inhibition of gastric emptying were about the same as their D_{50}'s for cholecystokinetic and pancreozyminic actions. In contrast, although both pentagastrin and heptadecapeptide gastrin inhibited gastric emptying, the doses required for this action were much higher than the D_{50}'s required for stimulation of gastric acid secretion. Both pure CCK and tryptophan were potent inhibitors of gastric emptying, and both protein secretion and gallbladder contraction were stimulated during tryptophan-inhibited gastric emptying. The results indicate that the inhibition of gastric emptying is attributable to CCK itself and not to an impurity in the CCK preparation. They further indicate that the inhibition of gastric emptying is one of the physiological actions of CCK, while the effect of gastrin is due to a pharmacological action.

5272 THE EFFECTS OF CHRONIC GANGLION BLOCKADE AND CHRONIC CHOLINESTERASE INHIBITION ON THE SENSITIVITY OF RABBIT STOMACH MUSCULARIS TO CHOLINERGIC AND ADRENERGIC AGONISTS. *(E.)* Lefever, G. S.; Green, R. D. (Univ. Illinois Med. Cent., Chicago). *J. Pharmacol. Exp. Ther.* 193(3):739-747, 1975.

The effects of chronic ganglion blockade and chronic cholinesterase inhibition on the sensitivity of the circular muscle from the rabbit stomach fundus to cholinergic and alpha adrenergic agonists were studied. The EC50 values, dissociation constants (K_A values), and efficacies of various agonists were determined in strips of stomach muscularis from control, chlorisondamine-pretreated and disulfoton-pretreated male and female rabbits. Strips from chlorisondamin-pretreated animals were supersensitive to carbachol but normosensitive to phenylephrine; the efficacy but not the K_A of carbachol was affected by this treatment. The dissociation constant (K_B) of atropine was unchanged. Muscle strips from animals treated with disulfoton were subsensitive to phenylephrine but normosensitive to amidephrine and carbachol, both the efficacy and K_A of phenylephrine appearing to be elevated by this treatment. The K_B of phentolamine remained unchanged. The results indicate that the chlorisondamine-induced supersensitivity is the result of a change beyond the level of the cholinergic receptors. The subsensitivity to phenylephrine after disulfoton pretreatment may result, at least in part, from a qualitative change in the alpha adrenergic receptors in the stomach muscularis.

5273 RADIOIMMUNOASSAY FOR MOTILIN. *(E.)* Dryburgh, J. R.; Brown, J. C. (Dep. Physiol., Univ. Br. Columbia, Vancouver, Canada). *Gastroenterology* 68(5):1169-1176, 1975.

A specific radioimmunoassay for the measurement of motilin in the sera of dogs was developed with the use of antisera to porcine motilin raised in guinea pigs. Antisera to the porcine motilin was raised in guinea pigs by multiple injections of both conjugated and unconjugated polypeptide. Alkalinization studies were then performed using sera from

fasted chronic dogs with extrinsically denervated pouches of the fundus of the stomach and Mann-Bollman fistulae into the duodenum. The radioimmunoassay procedure employed highly purified ^{125}I-motilin as the tracer; a sensitivity range of 10-320 pg was found. No cross-reactivity was demonstrated with gastric inhibitory polypeptide, secretin, glucagon, gastrin, cholecystokinin-pancreozymin, or vasoactive intestinal peptide. In the dogs with denervated pouches of the fundus of the stomach and Mann-Bollman fistulae, duodenal alkalinization resulted in an increase in the gastric motor activity in the fundic pouch with a corresponding increase in serum motilin, both parameters reaching their maxima within 5 min. These results strongly support the hypothesis that motilin is the peptide released upon alkalinization of the duodenum.

5274 PROPERTIES OF MECHANOSENSITIVE NEURONS WITHIN AUERBACH'S PLEXUS OF THE SMALL INTESTINE OF THE CAT. *(E.)* Mayer, C. J.; Wood, J. D. (Univ. Kansas Med. Cent., Kansas City). *Pfluegers Arch.* 357(1-2):35-49, 1975.

Extracellular techniques of single unit recording were used to study the response patterns of mechanosensitive neurons located within the ganglia of the myenteric plexus of the duodenum and jejunum of the cat. Segments of duodenum and jejunum from adult cats were mounted within chambers and the myenteric plexus was exposed. Action potentials of the mechanosensitive neurons within the ganglia were detected extracellularly, changes in the discharge pattern of the cell being elicited by mechanical distortion of the ganglion. Most recordings were from the periphery of the serosal side of the ganglia. Three different kinds of mechanosensitive neurons were detected within Auerbach's plexus. Neurons classified as fast-adapting mechanoreceptors discharged spikes at the onset of stimulation, and the discharge stopped during a sustained stimulus of constant intensity. Slowly-adapting mechanoreceptors maintained a steady discharge during sustained stimulation at a frequency which was a direct function of the intensity of the stimulus. Tonic-type neurons responded to mechanical stimulation with a prolonged train of spikes which had a consistent pattern from preparation to preparation. Once the tonic-type cells were triggered, the discharge followed a characteristic time course that was unchanged by further increase or decrease in stimulus activity. Tonic-type neurons are probably not first order sensory neurons, but they may be activated by input derived from primary mechanoreceptors. The frequency of discharge of the slowly-adapting mechanoreceptors was increased by histamine; this appeared to be secondary to histamine-induced contractile activity of the musculature.

5275 CONTROL OF GASTRIC EMPTYING BY OSMOLALITY OF DUODENAL CONTENTS IN MAN. *(E.)* Meeroff, J. C.; Go, V. L. W.; Phillips, S. F. (Mayo Clin., Rochester, Minn.). *Gastroenterology* 68(5): 1144-1151, 1975.

Osmoreceptors within the human duodenum were studied and some aspects of their mechanism of action were elucidated. A 500 ml liquid test meal of varying osmolality was introduced *via* gastric tube into 20 fasted male volunteers. Solutions of varying osmolality were also perfused into the duodenum *via* duodenal perfusion tube. Recovery of the nonabsorbable marker, phenol red, from the duodenum was used to measure and time the rate of gastric emptying. The rates of release of cholecystokinin-pancreozymin (CCK-PZ), gastrin, trypsin, and bile fluids were also measured. Gastric emptying was fastest when the duodenal contents were isotonic; nonisotonic duodenal contents (hypotonic and hypertonic) slowed emptying whether these conditions were achieved by nonisotonic duodenal perfusates or by emptying of a nonisotonic test meal. However, nonisotonic solutions in the stomach and jejunum did not slow gastric emptying so long as the duodenal contents were isotonic. Perfusion of the duodenum with 840 milliosmolar sodium chloride produced an insignificant increase in bile acid and trypsin outputs into the duodenum. When a test meal was given while hypertonic duodenal perfusion was continued, bile acid output peaked while the output of trypsin showed a concomitant sharp increase. When the test meal was emptied and its residual was aspirated from the stomach, the duodenal bile acid and trypsin values returned to basal levels. There were no changes in the fasting serum levels of gastrin or CCK-PZ. The results indicate that osmoreceptors, which slow gastric emptying, are present in the duodenum but not in the jejunum or stomach.

5276 EFFECT OF HYPOTHALAMIC, AMYGDALOID AND CINGULATE GYRUS STIMULATION ON COLONIC MOTILITY. *(Ukr.)* Motuznyi, V. A. (Inst. Physiol., State Univ., Kiev, USSR). *Fizol Zh* 19(6):753-759, 1973.

Colonic motility during different functional stages of the gastrointestinal tract was investigated together with the effect of electric stimulation of the hypothalamus, amygdala, and cingulate gyrus on this type of activity. Experiments were performed on dogs with colon fistulas, and four-polar electrodes implanted into the hypothalamus, amygdala, and cingulate gyrus. Data indicate that colonic motility is characterized by phases associated with mixing, intensive absorption of contents, formation of feces and their motion toward the rectum. The effects of electrical stimulation depended on the functional state of the colon and localization of stimulation. Stimulation of the ventromedial hypothalamic nuclei and macro- and microcellular basal amygdaloid nuclei increased colonic motility; stimulation of the posterior hypothalamus inhibited it. Electrical stimulation of the lateral hypothalamic nuclei affected colonic motility (causing contractions) only with current intensity of at least 1.2 millampere. The anterior cingular stimulation produced the proximal colonical motility followed by inhibition or relaxation in the distal colon.

5277 REASEC'S ACTION ON THE MOTORICITY OF THE ESOPHAGUS AND CARDIAC SPHINCTER IN CATS. *(Rus.)* Krasilnikov, K. B. (Inst. Physiol., Kiev. Univ., USSR). *Farmakol. Toksikol.* 38(2):200-202, 1975.

5278 STUDIES ON THE ROLE OF GASTRO-INTESTINAL INTEROCEPTORS IN THE PROCESS OF CONDITIONING OF ALIMENTARY INSTRUMENTAL REFLEXES. *(Pol.)* Cytawa, J.; Luszawska, D.; Schoenborn, R.; Zajec, M. (Inst. Med. Biol., Med. Acad., Gdansk, Poland). *Przegl. Lek.* 31(2):277-282, 1974.

5279 HORMONAL CONTROL OF GASTROINTESTINAL MOTILITY. *(E.)* Harvey, R. F. (Bristol R. Infirm., England). *Am. J. Dig. Dis.* 20(6):523-539, 1975.

5280 EFFECTS OF HEXAMETHONIUM AND OTHER GANGLIONIC BLOCKING AGENTS ON ELECTRICAL ACTIVITY OF THE ESOPHAGUS INDUCED BY VAGAL STIMULATION IN THE DOG. *(E.)* Toyama, T.; Yokoyama, I.; Nishi, K. (Kumamoto Univ. Med. Sch., Japan). *Eur. J. Pharmacol.* 31(1):63-71, 1975.

5281 ROLE OF PROSTAGLANDINS IN TONE AND EFFECTOR REACTIVITY OF THE ISOLATED RAT STOMACH PREPARATION. *(E.)* Frankhuijzen, A. L.; Bonta, I. L. (Dep. Pharmacol., Erasmus Univ., Rotterdam, Netherlands). *Eur. J. Pharmacol.* 31(1):44-52, 1975.

5282 CONTROL OF GASTRIC EMPTYING AND MOTILITY. *(E.)* Cooke, A. R. (VA Hosp., Iowa City, Iowa). *Gastroenterology* 68(4):804-816, 1975.

5283 MODIFICATION OF THE TONUS AND DRUG-INDUCED CONTRACTION OF THE ISOLATED RAT ILEUM BY REMOVAL OF Na FROM BATHING MEDIA. *(E.)* Taniyama, K. (Kobe Univ. Sch. Med., Japan). *Jpn. J. Pharmacol.* 24(3):433-438, 1974.

5284 ANALYSIS OF ERGOTAMINE--5-HT INTERACTION ON THE ISOLATED RAT STOMACH PREPARATION. *(E.)* Frankhuijzen, A. L. (Dep. Pharmacol., Erasmus Univ., Rotterdam, Netherlands). *Eur. J. Pharmacol.* 30(2):205-212, 1975.

See also, 5249, 5289, 5314, 5566.

SECRETION AND METABOLISM

5285 ACTION OF GASTRIN ON GASTROINTESTINAL STRUCTURE AND FUNCTION. *(E.)* Johnson, L. R.; Lichtenberger, L. M.; Copeland, E. M.; Dudrick, S. J.; Castro, G. A. (Univ. Texas Med. Sch., Houston). *Gastroenterology* 68(5, Part 1):1184-1192, 1975.

The effects of continuous pentagastrin (6 µg/kg-hr) and histamine (1 mg/kg-hr) infusion on gastrointestinal structure were studied in 12 male Sprague-Dawley rats maintained by i.v. hyperalimentation for 19 days. Compared to six sham-operated controls, i.v.-fed rats had a 3-fold lower antral and an 8-fold lower serum gastrin level; these levels were not affected significantly by continuous pentagastrin infusion. Oxyntic stomach, small intestine and pancreatic weights were reduced by 25%, 50% and 45%, resp., in hyperalimented rats. Weights of nongastrointestinal tract organs (kidneys, spleen and testes) were not affected by hyperalimentation. Pentagastrin infusion significantly increased oxyntic stomach, small intestine and pancreatic weights to levels near to or exceeding those of control, orally-fed animals. Pentagastrin infusion also completely prevented loss of brush border disaccharidase activity in hyperalimented rats. Infusion of histamine at a dose rate producing gastric acid secretion comparable to that produced by pentagastrin infusion failed to increase organ weights or disaccharidase activity in hyperalimented rats. It is thus concluded that pentagastrin prevents the changes in gastrointestinal structure and function caused by the absence of food in the gut and that the trophic action of gastrin is necessary for the maintenance of this functional and structural integrity.

5286 THE RELATIONSHIP OF INTESTINAL FAT TO AN AUGMENTED ACID AND GASTRIN RESPONSE IN DOGS WITH A REVERSIBLE PANCREATIC FISTULA. *(E.)* Hofmann, J. W.; Raih, T. J.; Go, V. L. W.; Wilson, S. D. (Med. Coll. Wisconsin, Milwaukee). *J. Surg. Res.* 18(4):409-415, 1975.

The relationship between duodenal fat and gastrin and acid responses to meals was investigated in mongrel dogs with reversible pancreatic fistulas and Heidenhain pouches. The acid response from the Heidenhain pouches and the serum gastrin response to a standardized meal were determined with the pancreatic fistula closed (pancreatic juice flowing normally into the duodenum) and open (pancreatic juices diverted externally). In a second group of dogs, the response to a test meal was determined during infusion of olive oil (0.38 cc/min) or oleic acid (0.23 cc/min) into the duodenum. The acid response to feeding with the fistula open was significantly greater than with the fistula closed, as was the mean 3-hr integrated gastrin response; the fasting serum gastrin concentrations with the fistula closed did not differ significantly from those with the fistula open. Infusion of either olive oil or oleic acid into the duodenum resulted in a significant inhibition of the meal-stimulated acid output when the pancreatic juices were allowed to drain normally into the duodenum; the acid response recovered more rapidly after olive oil perfusion than after oleic acid. With the fistula opened, only oleic acid infusion significantly inhibited the augmented acid response to a meal; the integrated gastrin response to a meal was not augmented. There were no pathologic changes noted in the pancreas or liver of any of the dogs. The results suggest that

the augmented acid and gastrin response to feeding that occurred when the pancreatic juice was diverted externally resulted from an impaired digestion of fats. They support the hypothesis that during a meal, the by-products of fat digestion normally stimulate the release of a hormone from the duodenum and small bowel and that this hormone acts, in part, to inhibit the meal-stimulated release of gastrin from the antrum.

5287 SIMULTANEOUS CHANGES IN PANCREATIC AND GASTRIC SECRETION INDUCED BY ACUTE INTRAVENOUS ETHANOL INFUSION: EFFECTS OF ATROPINE AND RESERPINE. *(E.)* Tiscornia, O. M.; Palasciano, G.; Dzieniszewski, J.; Sarles, H. (Unit Pathol. Dig. Dis., Marseille, France). *Am. J. Gastroenterol.* 63(5):389-395, 1975.

The effect of i.v. ethanol on gastric and pancreatic secretion was studied in dogs, along with the effect of simultaneous atropine or reserpine perfusion. Four dogs were prepared with chronic pancreatic fistulas and Thomas cannulas were inserted into the most dependent segment of the stomach. Four weeks later, the fasted dogs were subjected to continuous i.v. GIH secretin (0.5 CU/kg/hr) and gastrin (6 γ/kg/hr) infusion; i.v. ethanol infusion (1.3 g/kg) was begun when both the gastric and pancreatic secretion rates had reached plateau levels. In some experiments, atropine infusion (1.0 mg/hr) was begun after hormone infusion and before ethanol infusion, and in others, the dogs were treated with reserpine (0.10 mg/kg/24 hr) for 48 hr beginning 72 hr before hormone infusion. Acute i.v. ethanol inhibited the pancreatic secretion of protein (concentration and output) and stimulated gastric acid secretion. Atropine abolished the ethanol-mediated inhibition of pancreatic protein secretion but did not prevent the alcohol-mediated gastric acid stimulation. Reserpine did not modify the ethanol-mediated inhibition of pancreatic secretion, but it did induce a high plateau level of HCl secretion which obscured the presumed ethanol-mediated excitatory influences on the oxyntic cells. The results suggest that the ethanol-mediated inhibitory effects on pancreatic secretion are probably related to the stimulation of inhibitory fibers in the vagi. They also suggest that adrenoceptive receptors are involved in the excitatory effects of ethanol on HCl secretion.

5288 VAGOTOMY IN THE PREVENTION OF ULCERS AFTER PORTACAVAL SHUNT SURGERY IN RATS. *(Ger.)* Schumpelick, V.; Kauffmann-Mackh, G. (Surg. Clin., Univ. Hamburg, Germany). *Dtsch. Med. Wochenschr.* 99(38):1872-1877, 1974.

The effect of an end-to-side portacaval anastomosis on the incidence of stomach ulcers and the volume, pH, and total acidity of gastric juice was studied in male albino rats. The pylorus was ligated in all animals 14 days after shunt surgery or sham operations (laparotomy, clamping off of the vena cava and portal vein for 10 min, and ligation of the pyloric vein). The incidence of stomach ulcers and the area of these ulcers in mm^2/rat were significantly greater in animals with portacaval anastomoses than in sham-operated controls. The pH, volume, and total acidity of gastric juice were also significantly increased after shunt surgery. When rats underwent selective gastric vagotomy before pyloric ligation, neither rats with portacaval anastomoses nor controls developed gastric ulcers, but the pH of gastric juice was still significantly higher after shunt surgery than after sham operation. These findings suggest that development of ulcers in patients with portacaval shunts might be prevented by combining shunt surgery with selective gastric vagotomy.

5289 FURTHER OBSERVATIONS ON SOME GASTROINTESTINAL EFFECTS OF 3,3'-DIHYDROXYDIBUTYL ETHER. *(It.)* Impicciatore, M.; Chiavarini, M.; Bertaccini, G. (Inst. Pharmacol., Univ. Parma, Italy). *Ateneo Parmense [Acta Biomed.]* 45(1-2):17-28, 1974.

5290 CHANGES IN THE NITROGEN FRACTIONS OF CHYME IN THE STOMACH AND SMALL INTESTINE WHEN FOODS WERE ADMINISTERED IN VARIOUS PARTS OF THE GASTROINTESTINAL TRACT. *(Rus.)* Berkos, O. V.; Shedrunov, V. V. (I. P. Pavlov Inst. Physiol, Leningrad, USSR). *Biull. Eksp. Biol. Med.* 79(2):8-11, 1975.

5291 COMPENSATORY INCREASE IN CHOLINE ACETYLTRANSFERASE ACTIVITY IN SALIVARY GLANDS AND DIAPHRAGM MUSCLE OF THE RAT. *(E.)* Ekstrom, J. (Inst. Physiol., Univ. Lund, Sweden). *Acta Physiol. Scand.* 93(4):525-530, 1975.

5292 STUDIES ON 5'-NUCLEOTIDASE HISTOCHEMISTRY. III. 5'-NUCLEOTIDASE ACTIVITY IN SMOOTH MUSCLE CELLS OF THE RAT'S GASTROINTESTINAL TUBE. *(E.)* Klaushofer, K.; Pavelka, M. (Dep. Micromorphol., Univ. Vienna, Austria). *Histochemistry* 43(4):373-385, 1975.

5293 STUDIES OF ALBUMIN METABOLISM IN DOGS USING ^{51}Cr- and ^{125}I-LABELED ALBUMIN. *(E.)* Kallfelz, F. A.; Wallace, R. J. (New York State Vet. Coll., Cornell Univ., Ithaca). *Am. J. Dig. Dis.* 20(6):594-595, 1975.

5294 METHOTREXATE EFFECTS ON ENTERIC MUCOSA, LIVER AND PANCREAS OF NORMAL AND BLED RATS. *(E.)* Celle, G.; Dodero, M.; Pannacciulli, I.; Fresco, G.; Mansi, C.; Savarino, V.; Bogliolo, G. (Dep. Int. Med., Univ. Genova, Italy). *Eur. J. Cancer* 11(5):317-320, 1975.

5295 METABOLITES OF 5-^{3}H-CYCLOCYTIDINE IN MONKEYS GIVEN SINGLE INTRAVENOUS INJECTION. *(E.)* Hamada, F.; Sugihara, T.; Tsuyama, S.; Hirayama, H.; Kanai, T.; Nishimura, M.; Kuretani, K.; Hoshi, A. (Dep. Vet. Pharmacol., Univ. Tokyo, Japan). *Chem. Pharm. Bull.* 23(3):586-591, 1975.

5296 THE INTESTINAL PHASE OF GASTRIC SECRETION. *(E.)* Kester, R. C. (Ninewells Hosp., Dundee, Scotland). *Ann. R. Coll. Surg. Engl.* 56(5):231-245, 1975.

The kinetics of hormone release were studied following the application of a stimulus to the intestine, in order to pinpoint the time of maximum hormone concentration in portal blood for subsequent isolation of the hormone. Samples of portal blood were obtained from intestinally-fed donor dogs over 15 min, starting at the following intervals after the start of the meal: 15 min (11 animals); 30 min (17); 45 min (10) and 90 min (10). The animals were subjected to antrectomy, splenectomy, construction of a Heidenhain pouch, gastroduodenostomy, and a Roux-en-Y jejunocutaneous fistula. After 3 weeks, an end-to-side portacaval shunt was performed and an indwelling catheter was placed in the portal vein. To assay histamine activity, Heidenhain pouches were constructed in dogs and tests were performed every 4-6 weeks. Weanling pigs were provided with Heidenhain pouches and acid secretory responses were determined at 1/2 hr intervals for a control basal hour and for 4 hr after a standard oral meal. The intestinal phase hormone was released within 30 min of the application of a stimulus and rapidly reached peak concentration in the portal blood. I.v. infusion into a donor dog of active portal plasma from a shunted, intestinally-fed dog stimulated gastric acid secretion after a delay of approximately 1 hr, and required a mean of 1.5 hr to stimulate peak secretion, which suggests that intermediate steps may be necessary before the hormone can effectively stimulate the parietal cell mass. The pig developed portacaval shunt-related gastric acid hypersecretion in response to food comparable to that observed in the dog and in man. There was also a brief delay in gastric acid secretion after administration of crude intestinal extract, again indicating that some intermediate reactions must occur. In addition, it is suggested that pig jejunal mucosa is an appropriate source for isolation of the intestinal phase hormone.

5297 HEPATIC RESECTION AND GASTRIC SECRETION. *(E.)* Pinkerton, Jr., J. A.; Adkins, Jr., R. B.; Gobbel, Jr., W. G.; Foster, J. H. (Veterans Adm. Hosp., Nashville, Tenn.). *Am. Surg.* 41(6): 347-351, 1975.

Gastric acid secretion was studied in six dogs with Heidenhain pouches before and after 60-70% hepatectomy. In 5/6 dogs, hepatectomized gastric secretion was decreased 14-70% ($p<0.02$) following a high protein meal. One dog, which showed increased secretion, was found at autopsy to have a gastric stricture at the site of pouch construction. Conventional liver function tests were transiently abnormal in all dogs postoperatively. Autopsy revealed hyperplasia of remaining hepatic tissue but no gastric or duodenal ulceration in all dogs. These results indicate that hepatic tissue remaining after 60-70% hepatectomy is able to detoxify gastric secretagogues in the portal venous blood sufficiently so that gastric acid secretion is not affected.

5298 EFFECTS OF VAGOTOMY ON URECHOLINE MODIFIED HISTAMINE DOSE RESPONSES IN DOGS. *(E.)* Hirschowitz, B. I.; Hutchison, G. A. (Univ. Alabama Med. Cent., Birmingham). *Am. J. Physiol.* 228(5): 1313-1318, 1975.

Histamine incremental step-dose response studies were performed in nine dogs with gastric cannula and Komarov esophagostomy. Histamine (2-100 µg/kg-hr) in six successive 45 min steps was infused in four separate studies on each dog. In three dogs, step-dose responses were repeated after vagotomy. In both groups of dogs the dose responses were then studied against background i.v. infusions of urecholine (10, 20, 40, and 80 µg/kg-hr). Vagotomy acted as a competitive inhibitor (K_m doubled from 14 to 29 µg/kg-hr, while V_{max} was unchanged), and this effect could be reversed by <10 µg of urecholine/kg-hr. In both groups, increasing backgrounds of urecholine increased the V_{max} from about 14 meq of H^+/30 min to about 23-24 meq/30 min, and decreased histamine K_m from 14 µg/kg-hr to about 1-2 µg/kg-hr. Both changes were log linear. The effects of urecholine on histamine-stimulated H^+ secretion thus showed synergism (decreased K_m) and potentiation (increased V_{max}); the former was interpreted as a cholinergic effect on the parietal cell histamine receptors and the latter as the probable existence of spare cholinergic receptors not normally accessible to histamine. There were no intrinsic qualitative or quantitive differences in these responses between the vagotomized and innervated stomachs. These results support the use of vagotomy to reduce gastric secretion in patients with duodenal ulcer.

5299 SIGNIFICANCE OF GASTRIC SECRETORY CHANGES IN THE PATHOGENESIS OF STRESS ULCERS. *(E.)* Hase, T.; Anderson, P. R.; Mehlman, B. (Walter Reed Army Med. Cent., Washington, D.C.). *Am. J. Dig. Dis.* 20(5):443-449, 1975.

Gastric secretory patterns, plasma corticosterone levels and blood glucose concentrations were analyzed in fasted, rotationally stressed (60 rpm, 2 hr) male albino rats 3 hr after pyloric ligation. Gastric secretion was highest in controls, intermediate in stressed rats that developed ulcers, and lowest in stressed rats that did not develop ulcers. Neither high nor low gastric secretion correlated with stress-ulcer formation. When initial mucosal ischemia and secretory inhibition, which occurred in all stressed rats, were considered, those that developed ulcers manifested gastric hypersecretion when compared with those that did not develop ulcers. Mean plasma corticosterone was highest in stressed rats that developed ulcers, next highest in stressed rats that did not develop ulcers,

and lowest in control rats. Compared with normal rats, mean blood glucose was lowest in stressed rats and highest in controls. Hypoglycemic changes were more marked in stressed rats that developed ulcers than in stressed rats that did not develop ulcers. It is suggested that focal mucosal ischemic changes in the presence of hypoglycemia and resultant gastric hyperacidity favor ulcer formation in stressed animals.

5300 OXYGEN TENSION OF THE CANINE GASTRIC MUCOSA UNDER THE INFLUENCE OF VASOACTIVE AND ACID STIMULATING AGENTS AND FOLLOWING VAGOTOMY. *(E.)* Hartel, W.; Alken, P.; Linder, M. M. (Surg. Hosp. Infirm., Northwest Frankfurt am Main, Germany). *Bull. Soc. Int. Chir.* 33(5-6):417-421, 1974.

As a measure of microcirculation, oxygen tension was determined in male and female mongrel dogs under the influence of various pharmacologic agents and vagotomy. Fasted, anesthetized animals were subjected to a subdiaphragmatic total vagotomy or were injected i.v. with norepinephrine (2.5 and 5 µg/kg), naftidrofuryl (2-3 mg/kg), or pentagastrin (0.5-5 µg/kg). Central venous and arterial blood pressure and oxygen tension from the gastric antral mucosa were continuously recorded. I.v. norepinephrine caused a short but marked drop in the gastric mucosal oxygen tension, while naftidrofuryl increased the gastric mucosal oxygen tension in spite of a fall in the arterial blood pressure. Pentagastrin caused a temporary rise in oxygen tension followed by a more prolonged decrease. Vagotomy produced a moderate and more persistent drop in the mucosal oxygen tension. It is suggested that the naftidrofuryl-induced rise in mucosal oxygen tension was most probably caused by vasodilatation or, possibly, by an inhibition of cellular metabolism. The effects of pentagastrin can be explained in terms of an early vasodilatative effect, followed by a later onset of increased metabolic activity with high oxygen consumption.

5301 ADENYLATE CYCLASE AND PHOSPHODIESTERASE IN THE RAT GASTRIC MUCOSA AFTER STARVATION, FEEDING AND PENTAGASTRIN. *(E.)* Ruoff, H. J.; Sewing, K. Fr. (Pharmacol. Inst., Univ. Tubingen, Germany). *Naunyn Schmiedebergs Arch. Pharmacol.* 288(2-3):147-153, 1975.

The effects of starvation, feeding, and pentagastrin on the gastric mucosal activities of adenylate cyclase and phosphodiesterase were studied in female Wistar rats. The rats were killed after 24- or 48-hr starvation which was, in some cases, followed by a period of refeeding, or after a single i.v. injection of pentagastrin (125 µg/kg). The mucosa from the fundus of the stomach was removed and homogenized and assayed for adenylate cyclase and phosphodiesterase. Starvation for 24 or 48 hr reduced both the basal and NaF-stimulated activities of adenylate cyclase without affecting the activity of phosphodiesterase. Refeeding of starved rats slowly raised the adenylate cyclase activity to up to 430% of basal values within 4 hr; this effect was more pronounced under basal conditions than with NaF stimulation. Pentagastrin caused a stimulation of basal adenylate cyclase lasting 45 min; this was followed by a subsequent decrease in the basal and NaF-stimulated enzyme activity. Phosphodiesterase activity was not influenced by refeeding but was transiently inhibited by pentagastrin. The results indicate that the gastric mucosal cyclic AMP levels after starvation, feeding, and pentagastrin are regulated by adenylate cyclase, not phosphodiesterase. The rise in adenylate cyclase activity after refeeding appears to be related to functions other than H^+ and pepsin secretion.

5302 EFFECTS OF ETHANOL ON GASTRIC ACID SECRETION AND GASTRIC MUCOSAL CYCLIC AMP IN THE RAT. *(E.)* Puurunen, J.; Karppanen, H. (Dep. Pharmacol., Univ. Oulu, Finland). *Life Sci.* 16(10): 1513-1520, 1975.

The effect of ethanol on the secretion of gastric acid and on the content of cyclic AMP in the gastric mucosa was studied in male Sprague-Dawley rats. Ethanol was injected i.v. (10-800 mg/kg) or applied locally (1-10%) to the stomach for 5 or 25 min, after which gastric acid secretion was measured and the cyclic AMP content and activities of phosphodiesterase and adenyl cyclase in the glandular mucosa were determined. I.v. ethanol had no effect on the output of gastric acid, although there was a concentration-dependent decrease in acid secretion following local application. The latter effect was evident within 5 min; the rate of acid secretion decreased by about 30% with 1% ethanol and by 100% with 10% ethanol. Within 5 min of local application of 10% ethanol, the gastric mucosal content of cyclic AMP decreased by approximately 50%. A similar effect was observed *in vitro*; the decrease was about 30% with 2.5% ethanol, about 60% with 10% ethanol, and approximately 45% with 20% ethanol. In the presence of cyclic AMP (0.4-2µM), alcohol inhibited the activity of phosphodiesterase activity of the gastric mucosa in a competitive manner. The K_I-value was 0.16 M which would correspond to an alcohol concentration of 9.1%. In addition, ethanol caused a concentration-dependent inhibition of the activity of the gastric mucosal adenyl cyclase, the inhibition reaching nearly 100% with 0.166 M (9.4%) alcohol. It is concluded that the ethanol induced decrease of cyclic AMP in the gastric mucosa is due to a decreased formation of the nucleotide. The accompanying inhibition of acid secretion is consistent with the view that cyclic AMP is an intracellular regulator of gastric acid secretion. In view of the role of cyclic AMP in the control of cellular integrity, the ethanol-induced damage might also be at least partly due to the decreased mucosal concentration of cyclic AMP.

5303 PLASMA PROTEINS IN CANINE GASTRIC LYMPH. *(E.)* Bruggeman, T. M. (Dep. Physiol., Univ. Michigan, Ann Arbor). *Gastroenterology* 68(5): 1204-1210, 1975.

A method for obtaining samples of gastric interstitial fluid *via* the collection of gastric lymph is

described. Because it has not been possible to collect gastric lymph by the cannulation of lymph vessels, the methods of renal micropuncture were adapted to the collection and analysis of 0.5-3 µl samples obtained by puncturing lymph vessels on or immediately adjacent to the ventral surface of the dog's stomach. Samples of gastric lymph collected immediately after surgical exposure of the stomach were than subjected to acrylamide gel electrophoresis, and were found to show all the protein bands exhibited by simultaneously collected samples of plasma. Quantitative analysis of similar samples by cellulose acetate electrophoresis gave the following ratios of plasma:protein concentration in the gastric lymph: total protein, 0.51; albumin, 0.68; total globulins, 0.42; α_1-globulin, 0.49; α_2-globulin, 0.62; β-globulin, 0.35; fibrinogen, 0.39; and γ-globulin, 0.46. When samples were collected over 4-hr periods, the concentrations of all proteins in the lymph increased, a phenomenon which did not appear to be the result of increasing small pore size as the result of injury. When the mucosa was irrigated with solutions of dithiothreitol, a substance causing plasma shedding, the concentrations of proteins in the gastric lymph over a 4-hr period were similar to those found in control experiments. Gastric lymph mixed with 100 mN HCl was found to have proteolytic activity. The results suggest that albumin can leave gastric capillaries by restricted diffusion through small pores as well as by unrestricted diffusion through large pores.

5304 CHRONIC ALCOHOLISM AND ENDOGENOUS GASTRIN. *(E.)* Treffot, M. J.; Tiscornia, O. M.; Palasciano, G.; Hage, G.; Sarles, H. (Unit Res. Dig. Pathol., Marseille, France). *Am. J. Gastroenterol.* 63(1):29-32, 1975.

The effects of a meat meal with or without ethanol infusion on serum gastrin levels were studied in dogs chronically treated with ethanol. Six of 10 dogs with chronic pancreatic and gastric fistulas were given 50% ethanol (2.0 mg/kg) daily for 1 yr; the remaining animals were given no alcohol (controls). After an 18-hr fast, the dogs were fed 150 g of raw meat alone or in association with 50% ethanol. Blood samples were collected 5, 15, 30, 45, 60, 90, and 120 min later. After the meat meal alone, the gastrin blood levels increased more quickly and remained elevated longer in the dogs chronically treated with ethanol than in the control dogs; this difference reached a peak of 40% after 90 min. The gastrin blood values were also higher in the ethanol-treated dogs following the ingestion of meat with concomitant infusion of ethanol; this difference reached a maximum of 86% after 30 min. The percentage increase in serum gastrin in the nonalcoholic dogs was greater and longer lasting following the ingestion of meat alone. The results suggest that ethanol is effective in releasing gastrin from the antrum. They also suggest that chronic ethanol intoxication increases the storage of gastrin in and its release from the antrum, a phenomenon which would explain the increased parietal cell mass and gastric acid secretion shown by animals subjected to chronic alcohol intoxication.

5305 EFFECT OF VAGAL INNERVATION ON THE SECRETORY RESPONSES OF CANINE GASTRIC POUCHES EXPOSED TO BILE. *(E.)* Aubrey, D. A.; Burns, G. P. (Dep. Surg., State Univ. New York, Buffalo). *Am. J. Surg.* 129(5):518-522, 1975.

The secretory changes resulting from exposure of vagally innervated and vagally denervated canine fundic pouches to ox bile prior to a meat meal were studied. Male and female mongrel dogs were prepared with standard vagally denervated (Heidenhain) pouches or with similar pouches in which the anterior vagus nerve was preserved intact. Basal gastric secretions were measured in the fasted animals, after which a standard meat meal was administered and the gastric juice was collected in 15-min samples for 4 hr. In subsequent tests, the secretory responses of the pouches were assessed after preliminary exposure (30 min) to ox bile at pH 7 and pH 2. In the denervated pouches, the secretion patterns were similar after the meat meal alone (control) and after exposure of the pouches to bile at pH 7; however, the output of Na^+ was consistently higher in the pouches exposed to bile at pH 7 than in the control pouches. In the denervated pouches exposed to bile at pH 2, the output values for H^+, Cl^-, and K^+ were significantly lower than control values, while the output of Na^+ was significantly higher. The output of Na^+ and H^+ in the pouches exposed to bile at pH 2 was also significantly different from the output in the pouches exposed to bile at pH 7. The secretory volumes did not differ among the three groups. In the innervated pouches, the output of H^+ after exposure to bile at pH 7 was significantly reduced below control values and the output of Na^+ was significantly increased; these changes were more marked in the innervated pouches than in the denervated pouches. After exposure to bile at pH 2, the output of H^+ was more reduced in the secretions from the innervated pouches than in those from the denervated pouches, while the increase in Na^+ output was greater in the innervated pouches. The results suggest that the innervated mucosa is more susceptible than the denervated mucosa to the injurious effects of bile.

5306 EFFECT OF GASTRIC ALKALINIZATION ON LOWER ESOPHAGEAL SPHINCTER PRESSURE AND SERUM GASTRIN. *(E.)* Kline, M. M.; McCallum, R. W.; Curry, N.; Sturdevant, R. A. L. (Veteran's Admin. Wadsworth Hosp. Cent., Los Angeles, Calif.). *Gastroenterology* 68(5):1137-1139, 1975.

The effect of alkaline intragastric pH on the lower esophageal sphincter pressure (LESP) and on the serum gastrin concentration was measured in eight men without esophageal disease. After an overnight fast, four-lumen polyvinyl tube assemblies with four lateral orifices were fitted into the subjects. A 0.1 N $NaHCO_3$ solution (110 ml) or a control solution was administered *via* gastric instillation and the LESP and intragastric pH were measured both before and after administration. The serum gastrin concentration was also measured before and at 5, 10, and 20 min after administration. The mean basal LESP was 16.7 mm Hg and the mean basal gastrin concentration

was 21.6 fM/ml. Both parameters increased during administration of the solutions, but these increases were not significant; nor were there any significant differences between the LESP and serum gastrin measurements during instillation of the alkaline and control solutions. The gastric pH was greater than 7.0 during administration of the $NaHCO_3$ solution and between 2.0 and 6.9 during most of the control periods. It is concluded that intragastric alkalinization does not increase total radioimmunoassayable serum gastrin or LESP in man.

5307 INTERACTION OF CALCIUM AND MAGNESIUM ON GASTRIC ACID SECRETION AND SERUM GASTRIN CONCENTRATION IN MAN. *(E.)* Christiansen, J.; Rehfeld, J. F.; Stadil, F. (Bispebjerg Hosp., Copenhagen, Denmark). *Gastroenterology* 68(5):1140-1143, 1975.

The effect of magnesium on calcium- and pentagastrin-induced gastric acid secretion and on calcium-induced gastrin secretion were studied in healthy male and female volunteers. Levin tubes were placed in the subjects' stomachs and saline was administered i.v. for a control period of 1 hr. Calcium gluconate (30 ml/hr) or pentagastrin (0.5 µg/kg-hr) was infused i.v. over the next 3 hr; i.v. infusion of magnesium sulfate (30 mg/hr) was begun after 2 hr. Calcium gluconate increased the serum gastrin concentration as well as the gastric volume secretion, acidity, and acid output. The addition of magnesium sulfate to the infusate caused a slight but insignificant increase in serum gastrin concentration, whereas volume secretion, acidity, and acid output were significantly depressed. Magnesium sulfate had no effect on the gastric acid secretion induced by a submaximal pentagastrin infusion. These results indicate that magnesium antagonizes the activation of gastric acid secretion by calcium without suppressing gastrin release and may suggest that magnesium does not change the sensitivity of the parietal cell to gastrin.

5308 ACTION OF THE ANTI-INFLAMMATORY AGENTS, ACETYLSALICYLIC ACID, INDOMETHACIN AND FENOPROFEN ON THE GASTRIC MUCOSA OF DOGS. *(E.)* Lin, T. M.; Warrick, M. W.; Evans, D. C.; Nash, J. F. (Lilly Res. Lab., Eli Lilly Co., Indianapolis, Ind.). *Res. Commun. Chem. Pathol. Pharmacol.* 11(1):1-14, 1975.

The effects of acetylsalicylic acid (ASA), indomethacin, and fenoprofen on the integrity of the gastric mucosal barriers were measured by ionic fluxes into the gut lumen of three dogs with Heidenhain pouches. HCl (0.1 N, 30 ml) was infused into the pouches and removed at 30-min intervals for 6 hr. During the third and fourth periods, the acid instillate contained 4.5 mg/ml or 12.5 mM of ASA, indomethacin, or fenoprofen. All three agents affected the functional integrity of the mucosal barrier, but the characteristics of their actions differed. The H^+ concentration was decreased by ASA and fenoprofen and was increased by indomethacin, while all three drugs increased the net flux of Na^+, K^+, Ca^{++}, and Mg^{++}. Following initial augmentation of the instillate volume and H^+ concentration, indomethacin caused sustained back diffusion of acid from the lumen to the mucosa; this was accompanied by mucosal bleeding. The peak effects of ASA on the net ionic flux occurred during or immediately after drug infusion and were greater than those of indomethacin and fenoprofen. Fenoprofen at 12.5 mM had no effect on the concentrations of Ca^{++} or Mg^{++}, whereas both were significantly increased by equal molar doses of ASA or indomethacin. In onset of action, indomethacin was slow, but once disruption of functional integrity started, it continued for hours, showing no signs of returning to normal. In this sense, the total disruptive action of an equal weight or molar dose of indomethacin was greater than that of ASA or fenoprofen. At pH 1, the absorption of ASA from the Heidenhain pouch was greater than that of indomethacin or fenoprofen, their respective peak concentrations in the plasma being 53.9, 14.2, and 13.7 µM. The results suggest that fenoprofen may be less irritating and less ulcerogenic than ASA and indomethacin in the acid secreting stomach when the drugs are taken orally.

5309 EFFECT OF THE H_2-RECEPTOR ANTAGONISTS (BURIMAMIDE AND METIAMIDE) ON GASTRIC SECRETION STIMULATED BY HISTAMINE AND ITS METHYL DERIVATIVES. *(E.)* Preiss, D. U.; Code, C. F. (Mayo Clin., Rochester, Minn.). *J. Pharmacol. Exp. Ther.* 193(2):614-620, 1975.

The comparative effectiveness of burimamide and metiamide as antagonists of gastric secretion stimulated by histamine and its methyl derivatives, N^{α}-methylhistamine, N^{α},N^{α}-dimethylhistamine and 4-methylhistamine, was studied in dogs with vagally denervated (Heidenhain pouches) and vagally innervated gastric mucosal septal pouches (Pavlov pouches). The secretagogues were given by continuous i.v. infusion to produce steady states of secretory activity in the fasted conscious dogs; the antagonists were given by either rapid bolus i.v. injection or continuous i.v. infusion. With bolus injections, both antagonists promptly inhibited the secretion produced by histamine and its methyl derivatives. At similar control secretory rates, 40 µM/kg burimamide and 4 µM/kg metiamide produced the same degree of inhibition. When the antagonists were given by continuous i.v. infusion, the difference between them was greater, metiamide being 15-17 times more potent than buriamamide. The effectiveness of the antagonists was not changed by vagal denervation. Symptoms of toxicity to burimamide developed at doses in excess of 30 µM/kg/hr; none occurred with metiamide at 1.8-7.5 µM/kg/hr. In addition to establishing the greater potency of metiamide, the continuous i.v. infusion experiments revealed differences in the action of histamine and N^{α}-methylhistamine. With histamine providing the background secretion, the dose of buriamamide necessary to produce 50% inhibition was 14.7 times greater than that of metiamide; when N^{α}-methylhistamine was the agonist, the multiple was 16.7. The results suggest a stronger binding by N^{α}-methylhistamine than by histamine to the H_2-receptor and supports the contention that histamine and N^{α}-methylhistamine have different affinities for the H_2-receptor of the parietal cell.

5310 TOPICAL CALCIUM AND pH EFFECT ON GASTRIN RELEASE AND PARIETAL CELL SECRETION. *(E.)* McLaughlin, M. H.; Peskin, G. W.; Greenburg, A. G.; Saik, R. P. (Univ. Calif. Sch. Med., San Diego). *J. Surg. Res.* 18(4):427-430, 1975.

The effects of topical Ca^{++} and the pH effects of its delivery vehicle on antral gastrin release and parietal cell secretion were studied using the following solutions: $CaCl_2$ (pH 2 and pH 8, 300 mOsm); NaCl (pH 2 and pH 8, 300 mOsm). Integrated gastrin output and total acid output were calculated for each 45-min test period in the 36 cats studied. Both the fundus and the antrum were perfused with the test solutions to differentiate the effects of Ca^{++} and the pH changes on the system. Antral perfusion failed to consistently alter gastrin output regardless of pH or ion involved. No significant increase in acid production was noted after either Ca or alkaline stimulation of the antrum. Topical perfusion of the fundus with alkaline solutions caused increased acid secretion without elevated gastrin output, and Ca produced a significantly greater acid response than Na. It is suggested that direct topical and reflex effects on the parietal cell may play an important role in gastric acid secretion stimulated by ingestion of food and may be more significant than small physiologic changes in serum gastrin levels.

5311 EFFECT OF ATROPINE IN REDUCING PLASMA PROTEIN SHEDDING BY THE CANINE OXYNTIC GLANDULAR MUCOSA INDUCED BY TOPICAL IRRIGATION WITH HISTAMINE OR COBRA VENOM. *(E.)* Kauffman, Jr., G. L.; Davenport, H. W. (Dep. Physiol., Univ. Michigan, Ann Arbor). *Gastroenterology* 69(1):198-199, 1975.

The mucosa of a vagally denervated (Heidenhain) pouch of the oxyntic glandular area of the stomachs in two dogs was irrigated for 30 min with dodecyl sulfate (20 mM solution in 15 mM phosphate buffer, pH 7.4) to break the gastric mucosal barrier. The mucosa was then irrigated for 30 min with histamine (1 mg/ml) solution. The experiment was repeated six times, during three of which the dogs were given i.v. atropine (0.0125 mg/kg) 15 min before the histamine solution was applied. Plasma shedding was very much lowered when histamine irrigation was preceded by atropine administration. A vagally denervated pouch of another dog was irrigated once weekly with a solution of lyophilized venom of *Naja naja* (0.5 mg/ml in phosphate buffer, pH 7.4). At the fourth week the characteristic response to the venom occurred. For 8 weeks thereafter, irrigation with venom preceded by i.v. injection of atropine (0.1 mg/kg) was alternated with irrigation without atropine. Atropine greatly reduced, but did not abolish, plasma shedding induced by venom irrigation. Plasma shedding induced by topical irrigation with 30% v/v ethanol in phosphate buffer, pH 7.4, was not affected by i.v. atropine (0.0125 mg/kg). These data allow the conclusion that plasma shedding exhibits a cholinergic component.

5312 EFFECT OF THE ANTRUM AND VAGUS NERVE ON BETAZOLE STIMULATION OF THE RAT STOMACH IN PERFUSION EXPERIMENTS. *(Ger.)* Hantschmann, N. (Surg. Clin., Univ. Kiel, Germany). *Arzneim. Forsch.* 25(2):216-222, 1975.

In perfusion tests the effect of a single injection of betazole (50 mg/kg s.c.) was studied in fasting male Wistar rats which had undergone bilateral truncal vagotomy, vagotomy and pyloroplasty, or antrectomy. Unoperated rats served as controls, and the completeness of vagotomy was tested with insulin in all cases. When vagotomy was performed immediately before the perfusion test, no change occurred in the betazole-induced increase in gastric acid secretion. However, when vagotomy was performed at least four weeks before the perfusion test, betazole produced a very slow increase in acid secretion. Maximum values, attained 4 hr after injection, were half those obtained in unoperated controls and rats subjected to vagotomy immediately before the perfusion test. All but three of the 16 rats in this group had gastric dilatation and, despite a 24-hr fast, large quantities of food, hair, and dirt in their stomachs. Dilatation persisted, even after thorough rinsing of the stomach. To rule out the possibility that gastric dilatation affects acid secretion, truncal vagotomy was combined with pyloroplasty in 22 rats. Four or more weeks after surgery, betazole caused an increase in acid secretion which was just significantly above basal values within the first two hr after injection. Acid secretion then decreased, and basal values were reached 1 hr later. In 21 rats which had undergone antrectomy at least 4 weeks before the test, betazole produced a significant increase in acid secretion only 3 hr after injection, with maximum values being reached after 4 hr. When curves for rats subjected to vagotomy and pyloroplasty and those subjected to antrectomy alone were added, the resulting curves corresponded to those obtained on unoperated controls or on recently vagotomized animals during the first 3.5 hr of the perfusion test. These results suggest that betazole acts directly on the parietal cells and also produces cholinergic stimulation *via* the vagus nerve.

5313 COMPARISON OF THE EFFECT OF DIFFERENT FORMS OF A PROTEIN TEST MEAL ON GASTRIC ACID AND GASTRIN SECRETION. *(E.)* Ashby, D. B.; Himal, H. S. (Dep. Exp. Surg., McGill Univ., Montreal, Canada). *Am. J. Gastroenterol.* 63(4):321-323, 1975.

5314 DETERMINATION OF THE "BORDERLINE OF STASIS" IN PARIETAL CELL VAGOTOMY. PRELIMINARY EXPERIMENTAL STUDIES. *(E.)* Bone, J.; Brandsborg, O.; Brandsborg, M.; Mikkelsen, K.; Eriksen, P. O.; Amdrup, B. M. (Dep. Surg. Gastroenterol., Univ. Aarhus, Denmark). *Bull. Soc. Int. Chir.* 33(5-6):405-410, 1974.

5315 PHARMACOLOGICAL PROPERTIES OF N^1-PIPERONYL-N^4-3,7,11-TRIMETHYL-2,6,10-DODECATRIENYL-PIPERAZINE, A NEW NON-ANTICHOLINERGIC GASTRIC ANTISECRETORY AGENT. *(E.)* Bianchetti, A.; Guarneri, L.; Biraschi, L. M.; Riva, M. (Dep. Pharmacol., Res. Lab., Milan, Italy). *Arzneim. Forsch.* 25(4):580-589, 1975.

5316 PRODUCTION OF GASTRIC AND ESOPHAGEAL TUMORS IN RATS BY METHYLNITROSOCYANAMIDE, A POSSIBLE CANDIDATE OF ETIOLOGIC FACTORS FOR HUMAN GASTRIC CANCER. *(E.)* Endo, H.; Takahashi, K.; Kinoshita, N.; Baba, T. (Cancer Res. Inst., Kyushu Univ., Fukuoka, Japan). *Proc. Jpn. Acad.* 50(7):497-502, 1974.

5317 INHIBITION OF RELEASE OF PROSTAGLANDINS AS AN EXPLANATION OF SOME OF THE ACTIONS OF ANTI-INFLAMMATORY CORTICOSTEROIDS. *(E.)* Lewis, G. P.; Piper, P. J. (R. Coll. Surg. Engl., London, England). *Nature* 254(5498):308-311, 1975.

5318 AGE AT MATERNAL SEPARATION AND GASTRIC EROSION SUSCEPTIBILITY IN THE RAT. *(E.)* Ackerman, S. H.; Hofer, M. A.; Weiner, H. (Montefiore Hosp., Bronx, N.Y.). *Psychosom. Med.* 37(2):180-184, 1975.

5319 ANTIULCEROGENIC PROPERTY OF SODIUM POLYACRYLATE ON EXPERIMENTAL ULCERATIONS IN RATS. *(E.)* Kokue, E.; Hayama, T.; Nakamura, T. (Tokyo Univ. Agric. Technol., Japan). *Jpn. J. Pharmacol.* 24(4):621-626, 1974.

5320 THE IMPORTANCE OF THE STOMACH IN GASTRIN-INDUCED HYPOCALCEMIA IN THE RAT. *(E.)* Schulak, J. A.; Kaplan, E. L. (Dep. Surg., Univ. Chicago, Ill.). *Endocrinology* 96(5):1217-1220, 1975.

5321 CHOLECYSTOKINETIC POTENCY OF GASTRIN IN UNANAESTHETIZED CAT. *(E.)* Vagne, M.; Audigier, J. C. (INSERM U-45, Lyon, France). *Rend. Gastroenterol.* 6(3):168-173, 1974.

5322 THE NEGATIVE FEEDBACK MECHANISM OF GASTRIC ACID SECRETION: SIGNIFICANCE OF ACID IN THE GASTRIC JUICE IN MAN AND DOG. *(E.)* Itoh, Z.; Takeuchi, S.; Aizawa, I.; Honda, R. (Gunma Univ. Sch. Med., Maebashi, Japan). *Surgery* 77(5):648-660, 1975.

5323 SALBUTAMOL: INHIBITION OF GASTRIC ULCERATION IN THE RAT. *(E.)* Fielding, L. P.; Router, M.; Curwain, B. P. (St. Mary's Hosp. Med. Sch., London, England). *Br. J. Surg.* 62(2):162-163, 1975.

5324 DIBENAMINE BLOCKADE ON GASTRIC ACID SECRETION IN VITRO AND ITS PROTECTION BY HISTAMINE ANTAGONISTS. *(E.)* Watanabe, K.; Goto, Y. (Dep. Pharmacometrics, Res. Inst. Wakan-yaku, Japan). *Eur. J. Pharmacol.* 30(2):133-139, 1975.

5325 ROLE OF INTRA- AND EXTRAGASTRIC FACTORS IN ACIDIC SECRETION OF STOMACH. *(Ukr.)* Skliarov, Y. P. (Med. Inst., Lvov, USSR). *Fiziol. Zh.* 21(1):14-23, 1975.

5326 THE EFFECT OF VAGOTOMY ON THE MICROCIRCULATION IN THE WALL OF THE STOMACH. *(Rus.)* Lakhtina, V. P.; Kozlov, V. I. (II Moscow N. I. Pirogov Med. Inst., USSR). *Biull. Eksp. Biol. Med.* 79(3):1-128, 1975.

5327 STIMULATION OF THE STOMACHS OF RATS WITH PENTAGASTRIN AFTER VAGOTOMY IN THE PERFUSION TECHNIQUE. *(Ger.)* Hantschmann, N. (Surg. Clin., Univ. Kiel, Germany). *Arzneim. Forsch.* 24(12):2009-2015, 1974.

5328 THE EFFECT OF DIMETHYL 2(5-METHYL-4-IMIDAZOLYL)ETHYLAMINE(5-MeD) ON GASTRIC SECRETION IN THE CAT. *(It.)* Impicciatore, M.; Plazzi, V.; Mossini, F.; Chiavarini, M.; Bertaccini, G. (Inst. Pharmacol. Chem. Pharmacol., Univ. Parma, Italy). *Ateneo Parmense [Acta Biomed.]* 45(3):145-148, 1974.

5329 THE INFLUENCE OF VARIOUS ROUTES OF ADMINISTRATION ON THE EFFICACY OF THE GASTRIC INHIBITORY FACTOR IN THE RAT. *(Fr.)* Rouse, D.; Tetreault, L. (Hotel Dieu, Montreal, Canada). *Union Med. Canada* 104(6):970-975, 1975.

5330 PECULIARITIES OF CONDITIONAL AND UNCONDITIONAL REFLECTORY INFLUENCES UPON GASTRIC SECRETORY FUNCTION IN ELDERLY AND OLD PEOPLE. *(Ukr.)* Korkushko, O. V.; Kotko, D. N. (Acad. Med. Sci. USSR, Kiev, USSR). *Fiziol. Zh.* 21(1):57-62, 1975.

5331 THE ROLE OF GASTRINS IN PHYSIOPATHOLOGY OF THE STOMACH IN VIEW OF IMMUNOCHEMICAL STUDIES. *(Pol.)* Konturek, S. (Inst. Physiol., Med. Acad., Cracow, Poland). *Przegl. Lek.* 31(2):265-272, 1974.

5332 EFFECT OF METIAMIDE, AN ANTAGONIST OF HISTAMINE H_2-RECEPTORS, ON GASTRIC SECRETION. *(Pol.)* Konturek, S.; Demitrescu, T.; Radecki, T.; Dembinski, A.; Mitis, M. (Inst. Physiol., Med. Acad., Cracow, Poland). *Przegl. Lek.* 31(2):298-302, 1974.

5333 EFFECT OF CALCITONIN ON GASTRIC AND PANCREATIC SECRETION AND ON THE FORMATION OF EXPERIMENTAL PEPTIC ULCERS IN CATS. *(Pol.)* Radecki, T.; Konturek, S. J.; Thor, P.; Demitrescu, T.; Pucher, A. (Inst. Physiol., Med. Acad., Cracow, Poland). *Przegl. Lek.* 31(2):293-298, 1974.

5334 CYTOCHEMICAL AND ULTRASTRUCTURAL STUDY OF NONARGENTAFFIN GASTRIC ENDOCRINE CELLS OF THE RABBIT DURING ONTOGENESIS. *(Fr.)* Lefranc, G.; Pradal, G.; L'Hermite, A.; Dubin, J. C.; Tusques, J. (Fac. Med., Lab. Histol. Embryol., Nantes, France). *Ann. Histochim.* 19(3):157-172, 1974.

5335 ANTRAL CONTROL OF GASTRIC SECRETION. *(It.)* Monetti, G. (Univ. Bari, Italy). *Chir. Gastroenterol.* 7(2):220-257, 1973.

5336 ACUTE OVEREXERTION ON SECRETORY FUNCTION OF GASTRIC GLANDS. *(Ukr.)* Karpenko, L. N. (Med. Inst., Lvov, USSR). *Fiziol. Zh.* 21(1):41-44, 1975.

See also, 5207, 5213, 5232, 5272, 5287, 5590, 5609, 5610, 5798, 5800, 5848, 6489.

5337 ANALYSIS OF THE MECHANISM OF ACTION OF CALCIUM-INDUCED EXOCRINE PANCREATIC SECRETORY CHANGES IN THE DOG. *(E.)* Tiscornia, O.; Palasciano, G.; Dzieniszewski, J.; Verine, H.; Sarles, H. (Unit Res. Dig. Dis., Marseille, France). *Am. J. Gastroenterol.* 63(4):293-298, 1975.

To elucidate the role of the cholinergic system in the calcium-induced exocrine pancreatic secretory changes in the dog, an i.v. calcium infusion was superimposed on a continuous perfusion of secretin in dogs with and without an atropine-induced cholinergic blockade. GIH secretin (1 CU/kg/hr) was infused i.v. into mongrel dogs with gastric and duodenal fistulas (Thomas cannula); after 80 min, a second i.v. infusion of calcium (10 mg/kg) was begun and was continued for 60 min. In a second experiment, i.v. atropine infusion (1.5 mg/hr) was begun 80 min after the beginning of secretin infusion and calcium infusion was commenced 60 min later. In the absence of atropine-induced cholinergic blockade, i.v. calcium increased the flow rate, bicarbonate concentration and output, and protein concentration and output of the pancreatic juice; the greatest effects were on the protein concentration (increased by 148%) and output (increased by 173%). During atropine-induced blockade, i.v. calcium changed neither the flow rate nor the bicarbonate output of the secretin-induced pancreatic juice. On the other hand, i.v. calcium increased the protein concentration and output by 85% and 40%, resp., under these conditions. The results suggest that i.v. calcium enhances secretin-induced pancreatic secretion plateau values *via* two mechanisms: primarily by raising the cholinergic tone of the whole "pancreon"; and partially through a direct stimulus-secretion coupling effect on the acinar cells.

5338 MICROFILAMENTOUS SYSTEM AND SECRETION OF ENZYME IN THE EXOCRINE PANCREAS: EFFECT OF CYTOCHALASIN B. *(E.)* Bauduin, H.; Stock, C.; Vincent, D.; Grenier, J. F. (Fac. Med., Free Univ. Brussels, Belgium). *J. Cell Biol.* 66(1):165-181, 1975.

The influence of cytochalasin B (CB) on the exocrine function of the pancreas was studied *in vitro* using pieces of fasted adult rat pancreas. The pieces of pancreas were incubated in medium containing an L-amino acid mixture, D-glucose, buffer, and CB in ethanol solution. Parameters studied included the conversion of glucose into proteins and CO_2, the incorporation of glucose in the pancreatic pieces, sugar transport, the conversion of 2-deoxy-D-glucose to 2-deoxy-D-glucose-6-phosphate, calcium uptake, and the efflux of calcium. The pancreatic fragments were also studied by electron microscopy. The results showed that the microfilaments in the acinar cell are essentially located in the apical portion of the cell. Thin CB-sensitive microfilaments (50 Å) form the axis of the microvilli and a network lying beneath the apical membrane, while thicker filaments (100 Å), which are at least partly CB-insensitive, form bundles parallel to the plasma cell membrane and the desmosomal links. CB interaction with the acinar cell of the exocrine pancreas involves at least two sites: a membrane site involved in the inhibitory effect of CB on monosaccharide transport and a less sensitive site at the filamentous level which is at least partially responsible for the inhibitory effect of CB on the secretion of exportable enzyme from the pancreatic cell. CB did not alter the energy balance of the acinar cell or the exchanges of ^{45}Ca between the extracellular medium and the pancreatic tissue. CB at 2×10^{-7} and 2×10^{-6} M had secretagogue properties, whereas at 2×10^{-5} M, it had an inhibitory effect on stimulated secretion and secretagogue properties. The mechanism of these secretory effects is unknown. The data provide strong evidence for the involvement of the microfilamentous network in the last steps of the secretory cycle in the acinar cell of the exocrine pancreas.

5339 INCREASED PARATHYROID HORMONE SECRETION AND HYPOCALCEMIA IN EXPERIMENTAL PANCREATITIS: NECESSITY FOR AN INTACT THYROID GLAND. *(E.)* Norberg, H. P.; DeRoos, J.; Kaplan, E. L. (Univ. Chicago, Pritzker Sch. Med., Ill.). *Surgery* 77(6):773-779, 1975.

The role of the parathyroid and thyroid glands in the production of the hypocalcemia accompanying acute pancreatitis was studied in pigs. Acute pancreatitis was induced in thyroidectomized animals and in pigs with intact thyroids; sham-operated pigs served as controls. Before and at various intervals for 48 hr after the induction of pancreatitis, arterial blood samples were analyzed for blood urea nitrogen (BUN), serum electrolytes, total protein, amylase, total calcium, magnesium, phosphorus, and serum parathyroid hormone (PTH). The basal values for these parameters did not vary between normal, sham-operated, and thyroidectomized pigs. After induction of pancreatitis in the thyroid-intact pigs, however, serum amylase increased to more than 350% of the basal value, and serum calcium concentrations significantly increased between 8-24 hr, thereafter returning to normal. Serum PTH concentrations also increased significantly between 8-18 hr following the induction of pancreatitis, the peak PTH concentration (167% above basal values) occurring at 12 hr. Although serum amylase values also increased in the thyroidectomized pigs after the induction of pancreatitis, serum calcium and PTH did not vary from basal values. In all animals with acute pancreatitis, the serum magnesium, phosphorus, total proteins, BUN, and electrolytes remained within basal limits. The data support the view that calcitonin or another thyroid-related substance plays a role in the mechanism of the hypocalcemia which accompanies experimental pancreatitis in the pig.

5340 ACUTE HEMORRHAGIC PANCREATITIS (MASSIVE NECROSIS) WITH FAT NECROSIS INDUCED IN MICE BY DL-ETHIONINE FED WITH A CHOLINE-DEFICIENT DIET. *(E.)* Lombardi, B.; Estes, L. W.; Longnecker, D. S. (Univ. Pittsburgh Sch. Med., Pa.). *Am. J. Pathol.* 79(3):465-480, 1975.

The anatomicropathologic and ultrastructural alterations observed in the pancreatic tissues of mice fed DL-ethionine with a choline-deficient diet are described. Thirty-day-old female Swiss Webster mice were placed on choline-deficient diets (controls) or choline-deficient diets in which 0.5% DL-ethionine replaced an equal amount of sucrose (experimental animals). All experimental mice died within 5 days due to the development of an acute hemorrhagic pancreatitis with fat necrosis throughout the peritoneal cavity; none of the controls died within this time interval. The pancreatitis in the experimental animals was characterized by a massive necrosis of the exocrine parenchyma with intense hemorrhage and inflammatory reaction of the stroma. The sequence of histologic and ultrastructural alterations occurring in the acinar cells of the pancreas was studied in the controls and in mice fed the experimental diet for 1, 2, or 3 days. Pancreatic tissues of control mice were histologically normal. In those of mice fed the experimental diet for 1 day, acinar cells were intensely eosinophilic, and there were numerous zymogen granules and a considerable reduction in the amount of basal and perinuclear basophilia. After 2 days, the loss of basophilia was almost complete; there were numerous, small, cytoplasmic vacuoles, primarily in the basal portions of the cells; and the pancreatic stroma showed a moderate degree of edema and congestion. The vacuolation of the acinar cells became more extensive after 3 days and cellular necrosis was evident; the interstitial edema and congestion were more severe. The pancreatitis in the experimental mice appeared to be due to intraparenchymal activation of zymogens, resulting from a synergistic action of choline deficiency with the basic toxicity of ethionine toward pancreatic acinar cells. This experimental model closely simulates the acute hemorrhagic pancreatitis with fat necrosis occurring in humans and may prove useful for exploring the pathogenesis of this condition.

5341 FACTORS INVOLVED IN THE INTESTINAL FEEDBACK REGULATION OF PANCREATIC ENZYME SECRETION IN THE RAT. *(E.)* Schneeman, B. O.; Lyman, R. L. (Dep. Nutr. Sci., Univ. California, Berkeley). *Proc. Soc. Exp. Biol. Med.* 148(3):897-903, 1975.

The mechanisms involved in the intestinal stimulation of pancreatic secretion were studied using male Wistar rats, prepared with bile-pancreatic or pancreatic and intestinal cannulas. Lyophilized soybean trypsin inhibitor (SBTI), trypsin, diisopropyl fluorophosphate (DFP)-treated trypsin, chymotrypsin, or DFP-chymotrypsin was infused into the intestine; in other experiments, pancreozymin, trypsin, SBTI, chymotrypsinogen, or cholecystokinin (CCK) was infused into the jugular vein. The bile-pancreatic juice was collected during the infusion studies and was analyzed for total protein and chymotrypsin using the synthetic substrate *n*-benzoyl-L-tyrosine ethyl ester. SBTI stimulated pancreatic enzyme secretion only when introduced into the upper third of the small intestine. In rats whose pancreatic juice had previously been diverted for 10 hr, both trypsin and chymotrypsin suppressed the pancreatic protein output in a dose-dependent fashion, maximal suppression being obtained with 10 mg of trypsin/0.5 hr. DFP-trypsin and DFP-chymotrypsin had no such effect. The inhibitory effect of trypsin occurred only when the enzyme was infused into the upper third of the intestine. Trypsin, chymotrypsinogen, and SBTI had no significant effect on the pancreatic output when infused directly into the jugular vein. CCK infusion significantly increased the pancreatic enzyme output in rats whose pancreatic juice had been continually returned to the intestine; this increase was much smaller when the juice had been diverted for the preceding 10 hr. The results suggest that trypsin in the upper small intestine somehow prevents the release of CCK into the blood and that SBTI releases CCK by binding the trypsin and removing it from the intestine. Trypsin and chymotrypsin appear to exert their feedback regulation of pancreatic secretion in intestine.

5342 ENTEROPANCREATIC CIRCULATION OF DIGESTIVE ENZYMES. *(E.)* Liebow, C.; Rothman, S. S. (Cornell Univ. Med. Coll., New York, N. Y.). *Science* 189(4201):472-474, 1975.

The possible existence of a cycle involving the absorption of intact digestive enzymes by the intestine and their resecretion by the pancreas was investigated. Whole rabbit pancreases were excised and placed in an organ both containing bovine (^{3}H)chymotrypsinogen and human (^{131}I)albumin. The pancreatic duct was cannulated and the secretion was collected directly. The chymotrypsinogen concentrations in the secretion continued to rise for 1 to 2 hr before a steady-state secretory concentration was reached; about 3% of the chymotrypsinogen was of exogenous origin. The transpancreatic movement of this enzyme appeared to occur at least in part *via* an intracellular route. In another experiment, cholecystokinin-pancreozymin was added to the organ bath after (^{3}H)-labeled rabbit amylase (previously added) had achieved a steady-state concentration in the pancreatic secretion. After the addition of the hormone, the concentration of (^{3}H)amylase in the secretion rose markedly. In a further set of experiments, the mucosal to serosal flux of (^{3}H)chymotrypsinogen relative to that for (^{131}I)albumin was compared in gut sacs prepared from rabbit ileum. The permeability of the ileal membrane to chymotrypsinogen expressed per unit of concentration gradient was about 9-fold greater than that for albumin. Finally, to directly examine the circulation of enzyme, (^{3}H)chymotrypsinogen was placed in the intestine just down-stream from the intestinal opening of the pancreatic duct and the pancreatic secretion was collected from the cannulated duct. The ^{3}H chymotrypsinogen peaked in the secretion within 15 min; about 67% of the secreted label comigrated with bovine chymotrypsinogen under disc gel electrophoresis. The results indicate the existence of an enteropancreatic circulation for at least some digestive enzymes, although the magnitude of this process remains uncertain. In addition, the fluxes observed across the pancreas and intestine were of sufficient magnitude to indicate the possibility of a conservation mechanism.

5343 EFFECT OF OXYTETRACYCLINE AND CHLORAMPHENICOL ON THE EXOCRINE PANCREATIC FUNCTION IN THE RAT. *(E.)* Fleischer, K. (Med. Clin. Univ. Wurzburg, Germany). *Digestion* 11(5-6):382-391, 1974.

The effects of oxytetracycline and chloramphenicol pretreatment on pancreatic secretion under exogenous stimulation were studied in specific pathogen-free male rats of the Chbb: THOM strain. The animals were provided with bile-pancreatic cannulas and 12 days later were treated with oxytetracycline (20 mg/kg/hr, i.v. beginning 1 hr before the collection of pancreatic secretion, or 200 or 400 mg/kg/day, s.c. for 3 days prior to the collection of pancreatic secretion) or chloramphenicol (0.6 g/kg in 4% Tylose solution by stomach tube 12 hr before the collection of pancreatic secretion, or 0.6 or 1.2 g/kg/day for 3 days prior to the collection of pancreatic secretion). The serum antibiotic levels were determined in some animals. In others, pancreatic secretion was stimulated by the continuous i.v. infusion of 25 U/kg/hr of secretin and 25 U/kg/hr of pancreozymin. The protein and electrolyte concentrations and activities of amylase, trypsin, and lipase in the pancreatic secretion were measured. Short-term pretreatment with oxytetracycline or chloramphenicol produced no significant changes in the exogenously stimulated pancreatic secretion. However, after 3 days of pretreatment with either antibiotic, an approximately dose-dependent decrease in the exogenously stimulated pancreatic secretion was observed; this decrease was reflected in both the protein output and in the enzyme activities of the secretion. The volume and electrolyte output of the pancreatic secretion remained essentially unchanged. Immediately following the cessation of long-term antibiotic treatment, the serum oxytetracycline levels decreased steadily, while the serum levels of chloramphenicol decreased initially, and then remained relatively constant between 6 and 15 hr after treatment. Since short-term antibiotic treatment produced no changes in pancreatic secretion, the observed alteration after longer-term treatment appears to be largely attributable to a depression of enzyme protein synthesis.

5344 BIOCHEMICAL GENETICS OF α-AMYLASE ISOZYMES OF THE CHICKEN PANCREAS. *(E.)* Lehrner, L. M.; Malacinski, G. M. (Dep. Zool., Indiana Univ., Bloomington). *Biochem. Genet.* 13(1/2): 145-173, 1975.

α-Amylase isozyme patterns and patterns of inheritance were studied in pancreatic tissue from phenotypically different chickens. In the chicken population at large, three electrophoretically distinct pancreatic α-amylase isozymes were discovered. The isozymes were designated Pa 1, Pa 2 and Pa 3. The local population of chickens, however, possessed only isozymes Pa 2 and Pa 3 (present as three phenotypes: Amy_2 B, consisting of isozyme Pa 2, Amy_2 BC, consisting of isozymes Pa 2 plus Pa 3 and Amy_2 C, consisting of isozyme Pa 3). Pancreatic biopsy permitted the establishment of a breeding flock with defined amylase phenotypes. Matings of this flock established that amylases are inherited as codominant alleles at a single genetic locus. Further, there was no evidence of ontogenetic modification of the amylase isozymes. It was observed that amylase isozymes Pa 2 and Pa 3 each generated a family of at least three faster-migrating amylolytic proteins. These post-translationally-modifed amylases were designated Pa Xa, Pa Xb, and Pa Xc, where X represents the number of the progenitor amylase. Structural analyses of purified amylases demonstrated that all amylase isozymes are nonglycosidated, monomeric molecules of molecular weight 55,000. In addition, the data are consistent with the hypothesis that the faster-migrating amylases are produced by deamination of asparagine and/or glutamine residues.

5345 MORPHOLOGICAL AND FUNCTIONAL STUDY OF EXOCRINE PANCREAS WITH IMPAIRED INNERVATION. *(Rus.)* Eletskii, Iu. K.; Osman, A. Iu. (2nd Moscow Med. Inst., USSR). *Arkh. Anat. Gistol. Embriol.* 68(2):49-56, 1975.

5346 COMPARATIVE CYTOLOGICAL AND GENETIC ANALYSIS OF THE DIFFERENTIATION OF THE PANCREAS EPITHELIUM OF CERTAIN VERTEBRATES AFTER FREE AUTOTRANSPLANTATION. *(Rus.)* Gerlovin, E. Sh. (Leningrad Sanit. Hyg. Med. Inst., USSR). *Arkh. Anat. Gistol. Embriol.* 68(1):13-24, 1975.

5347 ISOPROTERENOL-INDUCED PANCREATIC SECRETION IN THE RAT; A COMPARISON WITH SECRETIN. *(E.)* Roze, C.; de la Tour, J.; Souchard, M.; Chariot, J.; Vaille, C.; Debray, C. (Hosp. Bichat, Paris, France). *Digestion* 11(5/6):458, 1974.

5348 CHRONIC ALCOHOL FEEDING AND GASTRIN PHASE OF RAT EXOCRINE PANCREATIC SECRETION. *(E.)* Cavarzan, A.; Teixeira, A.; Palasciano, G.; Dzieniszewski, J.; Tiscornia, O.; Sarles, H. (INSERM, Marseille, France). *Digestion* 11(5/6):442, 1974.

5349 OUTPUT OF NEWLY SYNTHETIZED PROTEINS BY HUMAN PANCREAS. *(E.)* Robberecht, P.; Cremer, M.; Deltenre, M.; Christophe, J. (Fac. Med., VLB, Brussels, Belgium). *Digestion* 11(5/6):457, 1974.

5350 EFFECT OF IONOPHORE A 23187 ON AMYLASE SECRETION AND PHOSPHOLIPID METABOLISM IN THE RAT EXOCRINE PANCREAS. *(E.)* Calderon, P.; Furnelle, J.; Winand, J.; Christophe, J. (Fac. Med., Univ. Libre, Brussels, Belgium). *Arch. Int. Physiol. Biochim.* 83(2):353-419, 1975.

5351 INTRACELLULAR ASPECTS OF THE PROCESS OF PROTEIN SYNTHESIS. *(E.)* Palade, G. (Yale Univ. Sch. Med., New Haven, Conn.). *Science* 189(4200): 347-358, 1975.

5352 PANCREATIC EXOCRINE SECRETORY PROTEINS. *(E.)* Greene, L. J. (Biol. Dep., Brookhaven Nat. Lab., Upton, N.Y.). *J. Surg. Oncol.* 7(2):151-154, 1975.

See also, 5197, 5208, 5229, 5287, 5294, 5333, 5425, 5455, 5574, 5609, 6047.

SECRETION AND METABOLISM

Hepatobiliary Tract

5353 BILE ACIDS SECRETION AND SYNTHESIS BY ISOLATED RAT HEPATOCYTES. *(E.)* Anwer, M. S.; Kroker, R.; Hegner, D. (Inst. Pharmacol., Toxicol. Pharm., Univ. Munich, Germany). *Biochem. Biophys. Res. Commun.* 64(2):603-609, 1975.

The synthesis and secretion of bile acids by isolated rat hepatocytes were studied. Hepatocytes were isolated from the livers of male Wistar rats and incubated in cell suspension. The supernatants were assayed for bile acids, lactate, pyruvate, and glucose and the results were compared with those obtained with bile samples collected *in vivo*. Bile acid secretion *in vitro* was linearly related to time for the first 3 hr of incubation; the net secretion rate was 23.2 mM/g of cells/min. The biliary excretion rate of bile acids by male Wistar rats has been reported to be 51.8 mM/g liver/min. The distribution of individual bile acids in the incubation media was significantly different from that in the *in vivo* bile samples. The ratio of taurine to glycine conjugates was significantly higher in the bile than in the incubation media; the ratio of dihydroxy to trihydroxy bile acids was significantly higher in the incubation media, and the ratio of chenodeoxycholic acid to cholic acid was higher in the incubation media than in the bile samples. The results indicate that isolated rat hepatocytes would provide a suitable experimental system for studying the effects of drugs and hepatic diseases on bile acid secretion and synthesis at the cellular level. The relatively higher synthesis of chenodeoxycholic acid in the hepatocytes could be used to study the mechanism controlling the relative proportion of primary bile acid synthesis from cholesterol.

5354 ANALYSIS OF REGULATORY FACTORS FOR UREA SYNTHESIS BY ISOLATED PERFUSED RAT LIVER. II. COMPARISON OF UREA SYNTHESIS IN LIVERS OF RATS SUBJECTED TO DIFFERENT DIETARY CONDITIONS. *(E.)* Saheki, T.; Tsuda, M.; Tanaka, T.; Katunuma, N. (Sch. Med., Tokushima Univ., Japan). *J. Biochem. (Tokyo)* 77(3):671-678, 1975.

The effects of dietary protein on urea synthesis and amino acid patterns in isolated, perfused rat liver were studied. Adult male Wistar rats were maintained for 4-24 days on 5% or 70% casein diets, after which their livers were perfused with medium with ammonium chloride as the nitrogen source. Urea formation in the livers of the rats fed the 70% casein diet was rapid and the efficiency of conversion of ammonia to urea was 97.9%. In contrast, urea formation in the livers of the rats fed the 5% casein diet was much slower and the efficiency of conversion of ammonia to urea was only 36.1%. In addition, the ratio of the rate of urea formation from ammonium chloride to the activity of ornithine transcarbamylase was much lower in the rats fed the low-protein diet than in those fed the high-protein diet. The addition of ornithine and *N*-acetylglutamate to the perfusate caused marked elevations in these ratios, two of which reached nearly the same level in both groups of rats. In the perfused livers from rats fed the 5% casein diet, a large portion of the ammonia added to the perfusate was fixed into an amino or an amide group of amino acids such as alanine, aspartate, and glutamine. In the perfused livers from the rats fed the 70% casein diet, most of the added ammonia was converted to urea. The results suggest that factors other than the amounts of urea cycle enzymes may play an important role in determining the flux through the urea cycle *in vivo*; these factors appear to be ornithine and *N*-acetylglutamate. The larger hepatic concentrations of nonessential amino acids in the rats fed the low protein diet may have resulted from the conversion of ammonia to nonessential amino acids; the fact that serine and glycine were not increased in the perfused livers suggests that their synthesis in the liver requires other factors or that they are converted rapidly into other compounds.

5355 METABOLISM OF RETINOL-BINDING PROTEIN AND VITAMIN A DURING HYPERVITAMINOSIS A IN THE RAT. *(E.)* Mallia, A. K.; Smith, J. E.; Goodman, D. S. (Columbia Univ. Coll. Physicians Surg., New York, N.Y.). *J. Lipid Res.* 16(3):180-188, 1975.

The effects of excess vitamin A on the plasma concentration and metabolism of retinol-binding protein (RBP) in the rat were studied. In the first experiment, weanling male Holtzman rats were given 0.14, 7.3, or 41 mg/day vitamin A, p.o., for 50-99 days. Plasma vitamin A and RBP levels were determined in samples obtained at various intervals between days 1 and 93. In the second study, adult Holtzman rats were given 0.14 or 34.4 mg retinal acetate daily for 23 days; these animals were also given daily p.o. doses of 0.67 μCi of ^{3}H-retinyl acetate. In both studies, administration of large, excessive doses of vitamin A resulted in substantial and significant decreases in the serum RBP levels. Excessive doses of vitamin A also produced fatty liver in association with a normal (in rats fed 7.3 mg vitamin A/day) or decreased (in rats fed 41 mg vitamin A/day) liver RBP concentration. In addition, the serum vitamin A levels were elevated in the animals fed

excessive vitamin A, an effect which was due primarily to large increases in the circulating retinyl ester levels. In the hypervitaminotic rats, most of the serum vitamin A and virtually all of the retinyl esters were found in association with the serum lipoproteins of less than 1.21 hydrated density. The results indicate that the serum lipoproteins play an important role in the transport of the vitamin A which accumulates in the serum during excessive vitamin A intake. It is suggested that the toxic manifestations of hypervitaminosis A occur when vitamin A circulates in the plasma and is presented to the membranes in a form other than bound to RBP. Plasma lipoproteins may nonspecifically deliver vitamin A to biological membranes, thus leading to vitamin A toxicity.

5356 COBALT STIMULATION OF HEME DEGRADATION IN THE LIVER: DISSOCIATION OF MICROSOMAL OXIDATION OF HEME FROM CYTOCHROME P-450. *(E.)* Maines, M. D.; Kappas, A. (Rockefeller Univ., New York, N.Y.). *J. Biol. Chem.* 250(11):4171-4177, 1975.

The relation of the cytochrome P-450 mixed function oxidase system to heme catabolism in liver cells was studied in rats. Male Sprague-Dawley rats were injected with cobalt chloride (60 mg/kg s.c.), cobalt protoporphyrin-IX or protoporphyrin-IX alone (4 μM/100 g i.p.), 3-amino-1,2,4-triazole (3 g/kg i.p.), or allylisopropylacetamide (400 mg/kg s.c.). In some cases, the animals also received i.p. injections of actinomycin D (0.06 mg/100 g or puromycin (2 mg/100 g). The rats were sacrificed after a 24-hr fast and liver microsomes analyzed for oxidative and δ-aminolevulinic acid synthetase activity. Cobalt chloride administration caused a marked increase in the oxidative degradation of heme (hematin, iron protoporphyrin-IX) beginning within 2 hr after cobalt treatment and reaching a maximum in 16-24 hr. During the rapid phase of stimulation, when heme oxidation was 450% above control values, there was a significant decrease in microsomal oxidative N-demethylation activity and in the microsomal heme content, with a nonsignificant decrease in the microsomal cytochrome P-450 concentration. Within 24 hr, the oxidative activity of the microsomal electron transport chain for drugs decreased to about 30% of control values and heme oxidation approached levels 800% above control. During this period there was a further decrease in the microsomal heme concentration, a significant decrease in cytochrome P-450, and an increase in the activity of δ-aminolevulinate synthetase. These parameters returned to normal within 72 hr. The effect of cobalt on heme oxidation could be inhibited by actinomycin D or puromycin administration; the effect could not be elicited by *in vitro* cobalt treatment of the microsomes nor attributed to any soluble cytoplasmic components. Cobalt protoporphyrin-IX was less effective than cobalt chloride in stimulating heme oxidation and protoporphyrin-IX alone had no effect. 3-Amino-1,2,4-triazole did not enhance hepatic heme oxidation while allylisopropylacetamide decreased it. The oxidative degradation of heme was not cytochrome P-450-dependent. The results exclude an obligatory role for cytochrome P-450 in the oxidation of heme compounds, although the possibility that this process is a heme-dependent oxidation is not ruled out.

5357 PREREPLICATIVE ENZYMIC CHANGES IN REGENERATING RAT LIVER. *(E.)* Ebina, Y.; Iwai, H.; Fukui, N.; Ohtsuka, H.; Miura, Y. (Chiba Univ. Sch. Med., Japan). *J. Biochem. (Tokyo)* 77(3):641-645, 1975.

The sequential changes in the activities of ornithine decarboxylase and inosinic acid dehydrogenase in the early period of hepatic regeneration were studied in rats, as was the activation of thymidine kinase and the decrease in cyclic-AMP-phosphodiesterase activity during the later phases of the G_1 period. Male Sprague-Dawley and female Donryu rats were subjected to partial hepatectomy and samples of the regenerating liver were examined for the various enzyme activities and the intracellular cyclic AMP level. The activity or ornithine decarboxylase and inosinic acid dehydrogenase increased soon after partial (70%) hepatectomy, the former continuing to increase for about 10 hr and remaining fairly high for about 32 hr, and the latter reaching a maximum at 8-12 hr. Thymidine kinase activity increased about 24 hr after surgery, with the regeneration process somewhat more rapid in the Sprague-Dawley strain than in the Donryu strain. The intracellular cyclic AMP level showed a biphasic increase at 8 and 24 hr, while the activity of cyclic AMP phosphodiesterase showed no appreciable change during the first hr then rapidly decreased from 16-18 hr. The low level of phosphodiesterase coincided with the period of the second steep rise in the cyclic AMP level. The results suggest that the increase in ornithine decarboxylase activity and the change in the intracellular level of cyclic AMP are important in the regulation of hepatic DNA replication. It is further suggested that inductions of inosinic acid dehydrogenase and thymidine kinase in the late prereplicative period in the regenerating liver is essential for DNA synthesis.

5358 THE KINETIC CHARACTERISTICS OF INHIBITION OF HEPATIC CHOLESTEROGENESIS BY LIPOPROTEINS OF INTESTINAL ORIGIN. *(E.)* Nervi, F. O.; Weis, H. J.; Dietschy, J. M. (Univ. Texas Health Sci. Cent., Dallas). *J. Biol. Chem.* 250(11):4145-4151, 1975.

The kinetic characteristics of the uptake of cholesterol from intestinal lipoproteins by the rat liver were studied and this phenomenon was correlated with the inhibition of hepatic cholesterogenesis by the lipoproteins. Lipoprotein extracted from donor rats was injected i.v. into female Sprague-Dawley-derived rats as a single bolus or by means of an infusion pump at a rate of 0.2 ml/min; in some cases, rats were instead fed a diet containing 0, 0.1, 0.3, 0.8, or 1.2% cholesterol. The animals were killed after 48 hr, the livers removed and sliced, and the rates of cholesterol and total ketone synthesis de-

termined. Net cholesterol uptake from the circulating intestinal lipoproteins took place only in the liver, and only this tissue manifested inhibition of cholesterol synthesis. Cholesterol uptake by the liver, quantified by a rise in the cholesterol ester content, was a linear function of time and of the amount of lipoprotein cholesterol administered to the animals. In all rats there was a general correlation between inhibition of the rate of cholesterol synthesis and the cholesterol ester content in the liver. However, there was no consistent quantitative relation between these two variables, suggesting either that cholesterol ester was not the immediate effector of the inhibition or, alternatively, that there was an intracellular localization of the effector at the site of control of the rate-limiting enzyme in the cholesterogenic pathway.

5359 ANALYSIS OF REGULATORY FACTORS FOR UREA SYNTHESIS BY ISOLATED PERFUSED RAT LIVER. I. UREA SYNTHESIS WITH AMMONIA AND GLUTAMINE AS NITROGEN SOURCES. *(E.)* Saheki, T.; Katunuma, N. (Sch. Med., Tokushima Univ., Japan). *J. Biochem. (Tokyo)* 77(3):659-669, 1975.

Urea synthesis in isolated, perfused Wistar rat liver was studied using ammonium chloride and glutamine as nitrogen sources. Samples of the perfusate were taken at 5-20 min intervals and analyzed for urea formation from ammonia and glutamine and for the additional influences of urea cycle intermediates and cofactors. Concentrations of urea, ammonia, and various amino acids were also measured in a sample of liver taken at the end of the experiment. The rate of urea formation increased with ammonium chloride concentration up to 5 mM but remained constant at concentrations of 5-20 mM. The concentration of ammonia in the medium needed to support the half-maximum velocity of urea formation was 0.7 mM. Aspartate (10 mM) and ornithine (2.5 mM) stimulated the rate of urea formation with ammonia. The greater part of the added ornithine was accumulated as citrulline in the liver, indicating that arginosuccinate synthetase was the rate-limiting enzyme of the urea cycle in the perfused liver. N-acetylglutamate also stimulated urea synthesis, but high concentrations (20 mM) were required for the maximum effect. Ornithine and N-acetylglutamate had an additive effect on urea formation. Urea formation from 10 mM glutamine occurred in 3 stages: in the first lag phase, a small amount of glutamine was removed from the medium; in the second stage, the glutamine level decreased rapidly and ammonia was accumulated in the perfusate; and in the third stage the glutamine concentration remained at a constant low level while the accumulated ammonia was rapidly converted into urea. The rate of urea formation in the third stage was much higher than with ammonia as the substrate. Urea formation from glutamine was also stimulated by the addition of ornithine, malate, or N-acetylglutamate to the perfusate. This stimulation was due to an effective conversion of ammonia to urea, the rate of removal of glutamine being unchanged.

5360 TEMPERATURE SENSITIVITY OF BROMOSULPHOPHTHALEIN CLEARANCE BY THE LIVER. *(E.)* Harris, P. J.; Noble, A. R.; Munday, K. A. (Dep. Physiol. Biochem., Med. Biol. Sci. Build., Southampton, England). *Experientia* 31(4):447-449, 1975.

The effect of temperature on BSP clearance by the isolated, perfused rat liver was studied. Livers from nonfasted male Wistar rats were perfused with a solution containing 10 mg BSP or 20 mM lactate and maintained at 37 C or 27 C. The bile duct was cannulated and the bile flow measured directly, and samples of the perfusate were analyzed at various intervals for BSP and lactate concentration. At 37 C, lactate was taken up from the perfusate at a mean rate of 0.84 μM/min/g, while at 27 C, the rate of lactate consumption was reduced by 45% to 0.46 μM/min/g. The mean rate of bile secretion was also reduced by 47% at 27 C (0.43 μliters/min/g) relative to 37 C (0.82 μliters/min/g). Changes in temperature had no effect on the structure of the liver cells. The initial mean rate of removal of BSP at 37 C was 21.7 μg/min/g, and at 27 C this was reduced to 7.4 μg/min/g; the percentage extraction of BSP at 37 C was 10.0%, while that at 27 C was 5.3%. In addition, while there was a linear disappearance of BSP from the perfusate at 27 C, the removal of BSP at 37 C was greater in the presence of high BSP concentrations than in the presence of low BSP concentrations in the perfusate. The correlation between the perfusate flow rate through the liver, as measured by application of the Fick principle to BSP clearance, and direct collation of the venous outflow was determined. The results indicate that under the conditions described, the mean hepatic blood flow rate can be calculated from BSP clearance at reduced temperatures.

5361 ALCOHOL SELECTION BY DBA AND C57BL MICE ARISING FROM OVA TRANSFERS. *(E.)* Randall, C. L.; Lester, D. (Sch. Med., Univ. Florida, Gainesville). *Nature* 255(5504):147-148, 1975.

To determine the relative contributions of heredity and environment to alcohol selection by mice, preferences and aversions to alcohol were measured in mice arising from ova transfers between mothers of different strains. Ova donors and recipients were primiparous adult C57BL/6 (alcohol preferrers) and DBA/2 (alcohol avoiders) mice; transfers were made between mice of the same and different strains. The pups were weaned at 25 days and housed individually for 7 days; during this period they were offered a choice between tap water and 7.5% ethanol. The C57BL mice weighed more and drank more alcohol and less water than the DBA mice, and control mice of both strains (not subjected to ova transfer) drank less alcohol and more water than the transferred mice. Thus, the C57BL offspring arising from ova transplanted into DBA mothers not only retained but increased the strain-typical high alcohol preference. The offspring arising from DBA ova transferred to C57BL mothers, on the other hand, also increased their alcohol consumption and did not retain their strain-typical aversion to alcohol. The results support a genetic basis for alcohol selection in the

C57BL mouse, the nature of which remains unknown, while demonstrating that alcohol avoidance in DBA mice is subject to environmental manipulation.

5362 EFFECT OF CHOLERETICS ON BILIARY AND URINARY EXCRETION OF CHOLECYSTOGRAPHIC AGENTS. *(E.)* Cooke, W. J.; Mudge, G. H. (Dartmouth Med. Sch., Hanover, N.H.). *Invest. Radiol.* 10(3):189-196, 1975.

The effects of the 2 choleretic agents, taurocholate and cinchophen, on the biliary and urinary excretion of 2 related cholecystographic agents, iopanoic acid and iophenoxic acid, were studied in adult mongrel dogs. The cystic duct was ligated and the common bile duct was cannulated in the anesthetized animals. Sodium iopanoate was infused at a constant rate of 1.6 ml/min in 2% mannitol in isotonic saline; iophenoxic acid was given in an initial dose of 4 mg/kg and sustaining infusions were not used. Sodium taurocholate was infused as a 1.3% solution in isotonic saline at 1.6 ml/min, and cinchophen was given in a priming dose of 50 mg/kg then infused in isotonic saline to provide 40 µg/kg/min. Urine was collected with an indwelling catheter, and liver biopsy samples were obtained during the experiment. The effect of each choleretic agent was specific for its related cholecystographic compound. Taurocholate increased both the biliary and urinary excretion of iopanoic acid and decreased its intrahepatic concentration, but had no effect on iophenoxic acid. Cinchophen had no effect on iopanoic acid but increased both the biliary and urinary excretion of iophenoxic acid. When examined against a preexisting high rate of bile flow, the active choleretic increased, rather than decreased, the biliary concentration of the respective cholecystographic compound. The increased excretion in both the bile and urine cannot be attributed simply to the reduced concentration gradient from plasma to bile, as has been previously suggested. The data are consistent with a direct action of the choleretic agent on hepatic mechanisms, either to decrease intracellular binding or to alter the characteristics of transport.

5363 OCTANOATE METABOLISM IN THE ISOLATED PERFUSED RAT LIVER. II. COMPARISON WITH A LONG CHAIN FATTY ACID. *(E.)* Bach, A.; Phan, T.; Metais, P. (Civil Hosp., Strasbourg, France). *Arch. Int. Physiol. Biochim.* 83(1):99-109, 1975.

The effects of octanoic acid and palmitoleic acid on hepatic metabolism were studied in the perfused livers of nonfasted male Wistar rats. The livers were perfused *in vitro* with lactic acid alone (10 mM/liter) or in combination with octanoic acid or palmitoleic acid (1.2 or 2.4 mM/liter). The pH was maintained constant at 7.40 and the neutralized supernatant was assayed at 0, 10, 20, 40, 60, and 80 min for lactate, pyruvate, β-hydroxybutyrate, acetoacetate, and glucose. Lactate alone significantly increased the concentrations of glucose, acetoacetate, and β-hydroxybutyrate at 80 min. When octanoate (1.2 mM/liter) was added to the perfusate, there was a nonsignificant decrease in pyruvate (40 min), a slight but significant increase in glucose (80 min), and a substantial increase in acetoacetate and β-hydroxybutyrate (80 min). When the concentration of octanoate was doubled, a significant decrease in pyruvate (40 min) and a substantial increase in β-hydroxybutyrate (80 min) were observed; the production of glucose and acetoacetate were not further increased. Addition of palmitoleate to the medium caused a significant increase in the pyruvate concentration (80 min) with no increase in glucose production. Octanoic acid was as ketogenic as palmitoleic acid when the 2 fatty acids were compared molecule by molecule, but the former was more ketogenic when the comparison was made on the basis of an equal number of acetyl groups. The results indicate that fatty acids do not play an important role in the rapid control of hepatic gluconeogenesis.

5364 THE RESPONSE OF THE RAT LIVER TO ALTERATIONS IN TOTAL PORTAL BLOOD FLOW. *(E.)* Weinbren, K.; Washington, S. L. A.; Smith, C. Y. (R. Postgrad. Med. Sch., London, England). *Br. J. Exp. Pathol.* 56(2):148-156, 1975.

The effects of changes in portal blood flow on the whole rat liver were studied. Male Wistar rats were subjected to a portacaval anastomosis, which left the liver entirely deprived of portal blood flow. After 1 week, the portal flow was reconstructed by ligating and dividing the portacaval anastomosis and re-anastomosing the portal vein to its original stump. The animals were fasted, injected i.p. with thymidine-6-^{3}H, and killed 24-72 hr later. Liver weights were lower in the rats subjected to portacaval anastomosis without reconstitution than in those in which the portal vein was reconstituted. In the animals bearing the anastomosis, there was a significant relative decrease in the weight of the anterior lobes compared with the posterior lobes; of the 2 posterior lobes, the right lobe weighed relatively more in relation to the omental lobe than in the control livers. Cell volumes were much reduced in the rats subjected to portacaval anastomosis, with values generally higher in the animals undergoing portal vein reconstitution than in those still bearing portacaval anastomoses. Specific activity of the liver DNA was also higher in the rats which had undergone reconstitution of the portal vein than in those in which the portacaval anastomosis was retained. Dye injection experiments indicated that the blood flowing through the reformed portal vein in the animals undergoing venous reconstitution reached all 4 liver lobes to a comparable degree. The results suggest that the predominant reaction to portacaval anastomosis was hypertrophy with a minor hyperplastic component. They support the view that the response after partial hepatectomy may represent a summation of reactions to different components of the rather complex stimulus engendered by that procedure.

5365 PLASMA CONCENTRATION OF LIPOPROTEIN-X (LP-X) IN EXPERIMENTAL BILE DUCT OBSTRUCTION. *(E.)* Ritland, S.; Bergan, A. (Univ. Hosp., Rikshospitalet, Oslo 1, Norway). *Scand. J. Gastroenterol.* 10(1):17-24, 1975.

Plasma levels of lipoprotein X were studied in bile duct-ligated mongrel dogs (six) with and without cholecystectomy. Dogs were divided into groups: dogs in groups I, IV, and VI had the common bile ducts ligated and cholecystectomy in the same session; dogs in groups II and V had bile duct ligation without cholecystectomy; dogs in group III had the common bile duct dissected free, but without bile duct ligation or cholecystectomy. Sequential quantitative determinations of lipoprotein X, measurements of plasma lipids, the initial rate of cholesterol esterification and liver function tests were performed. The lipoprotein appeared 11 to 21 hr after operation in the bile duct ligated and cholecystectomized dogs, and in dogs with preserved gallbladders 32 hr after operation. The lipoprotein levels were much higher in the cholecystectomized dogs throughout the observation periods (6-41 days) than in those with preserved gallbladders. Lipoprotein X concentration curves in plasma were biphasic in most of the bile duct-obstructed dogs. The first peak after 1-5 days was followed by a fall, and 10-14 days after operation another rise was observed. Fluctuations in the phospholipid and free cholesterol levels occured along with the changes in the lipoprotein. An inverse relation was determined between lipoprotein X and the activity of the lecithin: cholesterol acyltransferase ratio. It is suggested that the changes in plasma lipids during the first days following bile duct obstruction are initiated by abrupt reflux of phospholipids into plasma.

5366 PERSISTENT EFFECTS OF CHENODEOXYCHOLIC ACID ON BILIARY LIPIDS IN THE HAMSTER. *(E.)* Goldstein, L. I.; Bonorris, G. G.; Coyne, M. J.; Schoenfield, L. J. (Cedars-Sinai Med. Cent., Los Angeles, Calif.). *J. Lab. Clin. Med.* 85(6): 1032-1041, 1975.

The effects of chenodeoxycholic acid (CDC) ingestion on the biliary lipid composition, the rate-limiting enzymes involved in hepatic cholesterol and bile acid synthesis, and the synthesis of hepatic cholesterol and bile acids were studied in male Syrian golden hamsters to determine the mechanism and duration of the cholesterol desaturation action of CDC. The hamsters were given 20 mg/kg/day of CDC dissolved in 16% ethanol by feeding tube; controls were given the vehicle alone. The animals were killed at the end of the 30-day feeding period or at 10-day intervals thereafter, and the gallbladders and livers were removed for analysis. Administration of CDC for 30 days significantly increased the biliary bile acid and lecithin:cholesterol ratio, as well as the percentage of CDC in the bile. These effects persisted for 20 days after termination of CDC feeding but were not evident after 30 days. The hepatic HMG coenzyme A reductase and 7α-hydroxylase activities were significantly reduced by CDC; these effects persisted for 10, but not 20, days after the end of CDC treatment. CDC had no effect on hepatic cholesterol, although the hepatic CDC concentrations were significantly elevated throughout the experiment. The results suggest that CDC has a salutory effect on the biliary lipid composition while causing an increase in exogenous biliary CDC and a decrease in the endogenous synthesis of cholesterol. The persistence of decreased cholesterol synthesis and of improved biliary lipid composition after discontinuing CDC treatment provides a rationale for studying this effect in man and for testing intermittent CDC regimes for gallstone dissolution.

5367 ETHANOL-INDUCED CROSS TOLERANCE TO SEVERAL HOMOLOGOUS ALCOHOLS IN THE RAT. *(E.)* LeBlanc, A. E.; Kalant, H. (Addict. Res. Found. Ontario, Toronto, Canada). *Toxicol. Appl. Pharmacol.* 32(1):123-128, 1975.

Ethanol-induced cross tolerances to several lower aliphatic alcohols were studied in male Wistar rats. The animals were trained on the moving belt test which requires them to remain on a motor-driven belt which moves continuously over a shock-grid; if the animal touches the grid it receives a shock. The rats were then given a test dose of ethanol, *n*-propanol, *iso*-propanol, *n*-butanol, or *t*-butanol and tested on the moving belt, after which the ethanol-treated rats and a group of previously untreated rats were started on daily gavage with ethanol (6 mg/kg) for 20 days. At the end of this period, the rats were given test doses of ethanol and the other 4 alcohols and once again tested on the moving belt. The blood alcohol concentrations produced by the various doses of each alcohol were not significantly different in the pre- and postethanol groups; there was a linear correlation between dose and blood concentration in each case. This relation was not altered by chronic ethanol treatment. When the doses were corrected by the respective thermodynamic activity coefficients of the various alcohols, almost identical dose-response curves were obtained prior to chronic ethanol treatment. After chronic ethanol administration, the rats showed complete cross-tolerance to ethanol and the other alcohols; all of the corrected dose-response curves shifted to the same degree. This was not due to metabolic or distribution factors, which indicates that the findings reflect cross-tolerance in the nervous system itself. Thus, the effects of the various alcohols *in vivo* appear to depend on their common actions rather than on their unique actions, and involve complex integrative functions rather than the most basic membrane properties.

5368 ELECTRON MICROSCOPIC CYTOCHEMICAL CHARACTERIZATION OF BILE CANALICULI AND BILE DUCTS IN VITRO. *(E.)* Oda, M.; Phillips, M. J. (Toronto Gen. Hosp., Canada). *Virchows Arch. [Zellpathol.]* 18(2):109-118, 1975.

Subcellular fractions from liver homogenates were used for cytochemical analysis of Mg^{++} ATPase and 5'-nucleotidase (AMPase). Liver tissue from Wistar

rats was homogenized and the bile canaliculus-rich, and bile duct fractions isolated. Lead nitrate (200 mM) was used as the capture agent and 2 mM ATP or 5'-AMP as the substrate. After incubation, the suspension was centrifuged, fixed in glutaraldehyde, incubated, and postfixed in osmium tetroxide. Freshly prepared bile canaliculus or bile duct containing fractions were also prefixed in glutaraldehyde, recentrifuged, and postfixed in osmium tetroxide. Control fractions, prefixed or non-prefixed, were incubated without lead nitrate or substrate. Isolated bile canaliculi came through structurally intact, as did the bile ducts. The latter were usually surrounded by a basement membrane and partially disrupted hepatocytes. None of the controls showed electron-dense reaction products. Both prefixed and non-prefixed canaliculi showed Mg^{++} ATPase activity predominantly on the microvilli, though fine structural preservation was better in the prefixed specimens. Activity was also found on the canalicular microvilli of the partially disrupted hepatocytes, but not on the bile duct microvilli. The reaction products appeared to be deposited on the outer luminal surfaces of the canalicular membranes in the prefixed specimens. The non-prefixed canaliculi showed intense AMPase activity on the microvilli, and less intense activity on the lateral hepatic plasma membranes. There was a light reaction on vesicles presumably derived from fragmented plasma membranes. Demonstrable AMPase activity was dependent on duration of fixation. AMPase activity in the prefixed preparations was located primarily on the outer microvillar surfaces, and to a much lesser extent on the lateral plasma membranes and vesicles. No activity was seen on the microvilli or lateral or basal membranes of the bile ducts. No intracytoplasmic organelles showed activity of either AMPase or Mg^{++} ATPase. It is concluded that study of subcellular fractions is advantageous over study of whole liver tissue, and that the bile canalicular membrane is specialized for secretion of bile constituents from the cells into the canalicular lumens.

5369 THE EFFECTS OF ACUTE CADMIUM ADMINISTRATION IN THE LIVER AND KIDNEY OF THE RAT: LIGHT AND ELECTRON MICROSCOPIC STUDIES. *(E.)* Hoffmann, E. O.; Cook, J. A.; Di Luzio, N. R.; Coover, J. A. (Louisiana State Univ. Sch. Med., New Orleans). *Lab. Invest.* 32(5):655-664, 1975.

Rat liver and kidney cells were studied by light and electron microscopy after injection of cadmium acetate. Charles River rats were injected i.v. with 0.600 mg cadmium acetate/100 g 5% dextrose, *via* the dorsal vein of the penis. Controls were given dextrose solution only. For light microscopy, rats were killed at 4, 8, and 16 hr. Liver and kidney slices were fixed and stained in hematoxylin and eosin (6 μm sections) or Paragon multiple stain (1 μm sections). For electron microscopy, rats were killed at 8 hr, and fragments of liver only were fixed in paraformaldehyde-*s*-collidine buffer, postfixed in osmium tetroxide, and stained with uranyl acetate and lead citrate. Mortality was 0% at 4 hr, and 5% at 16 hr. Light microscopy showed insignificant changes in the kidneys. The liver appeared normal after 4 hr, but by 8 hr early lesions occurred in 50% of the rats, and at 16 hr, 90-95% showed scattered single parenchymal necrosis, congestion, and mild Kupffer cell vacuolation. There was a mild inflammatory cell infiltrate. Some rats dying before 16 hr showed necrosis, apparently in the mid-lobular zone. Electron microscope examination of Kupffer cells showed vacuoles, increase in dilated smooth endoplasmic reticulum, swelling of mitochondria, and cytoplasmic degradation. Desquamation was evident and polyribosomes were more prominent than in controls. Deposits of fibrin, platelets, cellular debris, inflammatory cells, and precipitated protein were found in the sinusoidal and Disse's spaces. Electron dense collagen fibers were observed in these areas. Scattered individual hepatocytes showed necrosis and areas of cytoplasmic degeneration. Many showed an increase in cytoplasmic vacuoles attributable to phagosomes, proliferation and dilation of smooth endoplasmic reticulum, pinocytotic vacuoles usually located toward the sinusoids and Disse's spaces, and lipid droplets. There was also detachment of ribosomes, fragmentation of membranes, and dilation of rough endoplasmic reticulum. Some cells showed more polyribosomes and loose ribosomes than controls, and occasional mitochondria were impregnated and swollen. Glycogen was consistently absent from hepatocytic cytoplasm. It is concluded that the lesions are similar to those produced by certain other non-metallic hepatotoxins.

5370 ANIMAL MODEL: CHOLELITHIASIS IN BRAZILIAN SQUIRREL MONKEYS (Saimiri sciureus). *(E.)* Lofland, H. B. (Bowman Gray Sch. Med., Winston-Salem, N.C.). *Am. J. Pathol.* 79(3):619-622, 1975.

Dietary factors in cholelithiasis were studied in the Brazilian squirrel monkey. Monkeys fed diets containing cholesterol and polyunsaturated fats for a period of nine months had almost 100% incidence of cholelithiasis. In monkeys fed cholesterol and safflower oil, 8/9 developed stones; in animals fed butter, 3/7 were affected; in those fed coconut oil, 1/7 was affected. Isotopic studies showed that the butter-fed animals had the largest bile acid pool size, and slowest turnover rate, while those fed safflower oil had the fastest turnover and the smallest pool size. Those fed coconut oil had a small pool size not differing significantly from the safflower oil-fed monkeys. The exacerbation of the disease by polyunsaturates in the monkeys resembles that in man. It is concluded that frequency of stone formation in the squirrel monkey is inversely related to the plasma cholesterol concentration, and provides a suitable model for the disease in man.

5371 BILIARY LIPIDS DURING VITAMIN C FEEDING IN HEALTHY PERSONS. *(E.)* Pederson, L. (Aalborg Hosp. North, Denmark). *Scand. J. Gastroenterol.* 10(3):311-314, 1975.

The influence of vitamin C on biliary lipids was studied in 10 healthy patients. None had taken vitamin C apart from dietary sources for at least one yr. Fasting duodenal bile samples were ob-

tained by duodenal intubation after cholecystokinin stimulation. Plasma samples were obtained simultaneously. During the following 10-15 days, each subject took 1 g vitamin C daily in divided doses; then fresh bile and plasma samples were obtained. Total bile acids, cholesterol and biliary phospholipids were determined. Percent concentrations of individual lipids in relation to total biliary lipids, the molar ratio between cholesterol and bile acids and the molar ratio between cholesterol and bile acids plus phospholipids were calculated. Differences between all values before and after treatment with vitamin C were statistically insignificant. It is concluded that vitamin C probably has no influence on biliary lipid composition in healthy humans.

5372 PROTEIN SYNTHESIS IN MITOCHONDRIA AND MICROSOMAL FRACTIONS FROM RAT BRAIN AND LIVER AFTER ACUTE OR CHRONIC ETHANOL ADMINISTRATION. *(E.)* Renis, M.; Giovine, A.; Bertolino, A. (Psychiatric Hosp., Casa Divina Provvidenza, Bisceglie (Bari), Italy). *Life Sci.* 16(9):1447-1458, 1975.

The effects of ethanol on the protein synthesizing capacity of the brain was studied in young male Wistar rats. Ethanol was administered to the rats for 20-25 days in the drinking water (20% ethanol) or was administered p.o. by stomach tube (5 g/kg) 3 hr before sacrifice. Incorporation of ^{14}C- or ^{3}H-leucine into brain and liver microsomes and mitochondria was determined *in vivo* and *in vitro*. For the study of protein synthesis *in vivo*, rats were sacrificed 1/2 hr after i.p. injection of ^{3}H-leucine. Protein synthesis in the mitochondrial preparations was significantly inhibited by chloramphenicol and practically unaffected by cycloheximide; the latter, however, specifically inhibited the microsomal preparations. Chronic ethanol administration increased *in vitro* protein synthesis in liver microsomes and mitochondria, while acute ethanol treatment inhibited it. Ethanol administration had no significant effects on protein synthesis in brain mitochondria, although chronic treatment had an inhibitory effect on brain microsomal preparations. The ethanol effect on liver microsomes was noticeably extended at the ribosomal level, as indicated by measurements of ^{3}H-leucine incorporation in isolated microsomes crossed with pH 5 enzymes from rat liver and brain after chronic ethanol treatment. Acute ethanol treatment decreased the *in vivo* incorporation of radioactive leucine into liver mitochondria and brain and liver microsomes, with no significant effects on brain mitochondria. Chronic administration had no significant effects on *in vivo* protein synthesis. These results indicate that ethanol affects protein synthesis *in vitro* only after chronic administration; the effects are opposite in the brain and liver but similar in the microsomal and mitochondrial fractions of each organ.

5373 ISOLATION OF CENTROLOBULAR AND PERILOBULAR HEPATOCYTES AFTER PHENOBARBITAL TREATMENT. *(E.)* Wanson, J. C.; Drochmans, P.; May, C.; Penasse, W.; Popowski, A. (Lab. Cytol. Exp. Oncol., Free Univ., Brussels, Belgium). *J. Cell Biol.* 66(11):23-41, 1975.

I.p. phenobarbital injections (100 mg/kg/day for 3-7 days) were used to induce a clear selective response in the centrolobular hepatocytes of adult female Sprague-Dawley rats. Following phenobarbital treatment, the hepatocytes were examined by electron microscopy, and their protein, DNA, RNA, and glycogen concentrations and glucose-6-phosphatase activities determined. The phenobarbital injections induced an intense proliferation of the smooth endoplasmic reticulum (ER) associated with a decrease in the activity of glucose-6-phosphatase. The centrolobular hepatocytes were the first affected, leading to an enhancement of liver lobule heterogeneity. Two liver cell populations were distinguished following subfractionation of the hepatocytes on Ficoll density gradients: light hepatocytes (mean density 1.10), which had the same morphological characteristics as the centrolobular cells, i.e., an abundant smooth ER composed of tubular elements, numerous small mitochondria, and few glycogen particles; and heavy hepatocytes (mean density 1.14), which were characterized by large and compact glycogen areas and prominent rough endoplasmic cisternae, as are the perilobular cells. After incubation in Wachstein-Meisel medium, the centrolobular hepatocytes exhibited dispersed reaction sites of glucose-6-phosphatase activity, whereas the periolobular cells showed a continuous and intense reaction pattern. Morphometric determinations showed the centrolobular phenobarbital hepatocytes to be considerably enlarged (mean diameter: 23.7 μm); the perilobular hepatocytes had a significantly smaller mean diameter of 19.2 μm, which is close to the values of control liver cells.

5374 COLCHICINE INHIBITION OF PLASMA PROTEIN RELEASE FROM RAT HEPATOCYTES. *(E.)* Redman, C. M.; Banerjee, D.; Howell, K.; Palade, G. E. (New York Blood Cent., N.Y.). *J. Cell Biol.* 66(1): 42-59, 1975.

The effects of colchicine and functionally related agents on the *in vitro* and *in vivo* secretion of albumin and other plasma proteins by the rat liver were studied. Ethanol (0.6 g/100 g of a 50% solution) was given p.o. by stomach tube to fasted male rats, after which colchicine or radioactive amino acid was administered i.v. *In vitro*, rat liver slices were incubated with radioactive leucine, washed, and transferred to a "chase" medium containing nonradioactive amino acids and colchicine or vinblastine. Radioactivity in the albumin and other plasma proteins was determined by immunoprecipitation, and liver samples from the rats treated *in vivo* were examined by electron microscopy. Colchicine inhibited the secretion of albumin and other plasma proteins both *in vitro* and *in vivo*. *In vitro* secretion of these proteins by the rat liver slices was inhibited by doses as low as 10^{-6} M, the maximal effect occurring at 10^{-5} M. Inhibition of secretion was accompanied by a concomitant retention of nonsecreted proteins within the slices. Colchicine did not inhibit protein synthesis at these concentrations. Vinblastine also inhibited plasma protein secretion, while lumicolchicine, griseofulvin, and cytochalasin B had

no such effect. The inhibition of secretion by colchicine and vinblastine was not due to changes in the intracellular nucleotide phosphate levels. When administered i.v. at 10–25 μM/100 g, colchicine acted within 2 min and its inhibitory effect lasted for at least 3 hr. This agent had no effect on the transport of secretory proteins in the rough or smooth endoplasmic reticulum, but it caused these proteins to accumulate in the Golgi-derived secretory vesicles. Neither the concentration nor the arrangement of microtubules within the Golgi regions appeared to be affected by colchicine at the concentrations used. The results indicate that colchicine acts by hampering the discharge of secretory proteins from Golgi-derived secretory vacuoles into the space of Disse.

5375 ISOLATION AND SUBFRACTIONATION ON FICOLL GRADIENTS OF ADULT RAT HEPATOCYTES: SIZE, MORPHOLOGY, AND BIOCHEMICAL CHARACTERISTICS OF CELL FRACTIONS. *(E.)* Drochmans, P.; Wanson, J. C.; Mosselmans, R. (Lab. Cytol. Exp. Oncol., Free Univ., Brussels, Belgium). *J. Cell Biol.* 66(1):1-22, 1975.

A technique for the isolation and subfractionation of adult rat hepatocytes is described. The recirculating perfusion of rat liver with a Ca^{++}-free Hank's solution produced a release of the adhesiveness of the cells and a cleaving of the desmosomes. The subsequent addition of collagenase and hyaluronidase to the perfusion medium led to a complete dissociation of the liver tissue into a mixture of isolated cells and cell cords in which the hepatocytes remained connected with specific junctional differentiations, namely the gap and tight junctions. Individual cells were released by submitting the suspension of cell trabeculae to a gentle rolling. The gap junctions were thereby ruptured, generally remaining attached to one cell and taking with them a small portion of cytoplasm. In this manner, 60–65% of the liver parenchymal cells were isolated; endothelial cells and other cells of the connective tissue were not recovered. The ultrastructural preservation of the isolated hepatocytes was excellent and the glucose-6-phosphatase activity appeared unaltered in most cases. Protein, DNA, and RNA recovery in the preparations was also satisfactory, amounting to 70% of that found in liver homogenate; glycogen was partly lost or degraded during the manipulations. Measurements of cell diameter confirmed the preservation of the original volume of the *in situ* hepatocytes and the presence of more than 1 type of parenchymal cell. By submitting this heterogeneous cell population to an isopycnic density gradient centrifugation, 2 types of hepatocytes were distinguished: the light hepatocytes, with a mean diameter of 20.5 μm and a mean density of 1.10, were characterized by an extended smooth endoplasmic reticulum entrapping dispersed α-glycogen particles; the heavy hepatocytes, with a mean diameter of 19.0 μm and a mean density of 1.14, presented a relatively reduced compartment of smooth endoplasmic reticulum and large accumulations of glycogen. It is suggested that the low-density cell fraction corresponds to centrolobular cells and the high-density fraction to periolobular hepatocytes.

5376 IMPAIRED HEPATIC RELEASE OF TRIGLYCERIDES: A POSSIBLE CAUSE OF ACUTE ALCOHOLIC FATTY LIVER IN THE RHESUS MONKEY. *(E.)* Nair, C. R.; Nirankari, O. P.; Chhuttani, P. N.; Datta, D. V. (Postgrad. Inst. Med. Educ. Res., Chandigarh, India). *Clin. Sci. Mol. Med.* 48(5):453-455, 1975.

The effects of Triton WR-1339 on the plasma triglycerides of ethanol-treated Rhesus monkeys were studied to determine the mechanism involved in the development of acute alcoholic fatty liver. Well-nourished Rhesus monkeys with no histological evidence of liver disease received an infusion of sodium chloride for 30 min followed by an i.v. infusion of Triton WR-1339 (20% solution at a rate of 26.6 mg/min/kg for 10 min). In some animals, the Triton WR-1339 infusion was preceded by a 30-min i.v. infusion of ethanol (214 mM/liter in saline at a rate of 1.8 mM/min/kg). Blood samples were collected 0, 2, 4, and 6 hr after the end of the Triton infusion, and the plasma triglycerides, plasma Triton, and blood ethanol levels were estimated. Liver biopsy tissue was also examined histologically and the liver triglycerides measured. In addition, the inhibitory effect of Triton on plasma lipoprotein lipase was studied *in vitro*. In the saline-Triton group, the plasma triglycerides rose 10- to 19-fold over basal values by the sixth hr; the liver triglyceride levels remained normal. In contrast, the ethanol-Triton group showed only a 1.2- to 2.3-fold rise in plasma triglycerides, with a significant rise in the liver triglycerides. Ethanol had no significant effect on the plasma Triton concentration, and inhibition of lipolysis was 94% in the saline-Triton group compared with 93.5% in the ethanol-Triton group. It is suggested that the reductive effect of Triton on the plasma triglycerides following ethanol administration is due to a reduced release of triglyceride from the liver. Acute alcoholic fatty liver in Rhesus monkeys is probably due to impairment in the hepatic release of triglycerides.

5377 EFFECT OF CHRONIC INTAKE OF ETHANOL ON LACTATE/PYRUVATE AND β-HYDROXYBUTYRATE/ACETOACETATE RATIOS IN RAT LIVER. *(E.)* Khanna, J. M.; Kalant, H.; Loth, J. (Dep. Pharmacol., Univ. Toronto, Canada). *Can. J. Physiol. Pharmacol.* 53(2):299-303, 1975.

Lactate/pyruvate and β-hydroxybutyrate/acetoacetate ratios were measured in male Wistar rats chronically treated with ethanol. The experimental rats were fed a liquid diet providing 35% of the calories as ethanol, while pair-fed controls received a similar diet in which the alcohol was replaced by an equicaloric concentration of sucrose. The ethanol diet was discontinued immediately, 24 hr, or 48 hr before sacrifice (1 month after the beginning of the experiment). In some animals in which the ethanol feeding was terminated 48 hr prior to sacrifice, and in their controls, ethanol (1 g/kg) was injected i.p. 1 or 3 hr before sacrifice. The lactate/pyruvate ratio was significantly higher in the ethanol-fed rats than in the controls when the ethanol diet was continued until sacrifice, but the two groups did not differ in this respect when the ethanol diet was discontinued prior to sacrifice. The β-hydroxybutyrate/

acetoacetate ratio was also significantly higher in the ethanol group when the ethanol diet was continued until the time of sacrifice; again, however, this difference disappeared when the ethanol feeding was discontinued 24 or 48 hr prior to sacrifice. In contrast, when ethanol was injected 1-3 hr before sacrifice, the β-hydroxybutyrate/acetoacetate ratio was significantly higher in the controls. The results are compatible with an adaptive increase in the mitochondrial reoxidation of NADH in the chronic alcohol groups, but the possibility of a change due to alcohol withdrawal cannot be excluded.

5378 ON THE QUANTITATIVE STEREO-MORPHOLOGY OF MICROBODIES IN RAT HEPATOCYTES. *(E.)* Wedel, F. P.; Berger, E. R. (VA Hosp., Brooklyn, N.Y.). *J. Ultrastruct. Res.* 51(2):153-165, 1975.

Microbodies in rat hepatocytes were studied quantitatively, stereologically, morphometrically, and ultrastructurally. Employing the modified Sjostrand technique for 3-dimensional reconstructions from serial thin sections for electron microscopy, two populations of particles were revealed. The larger class P microbodies were spherical particles, about 410 nm in diameter, and bounded by a limiting membrane about 110 Å thick; they contained a finely granular matrix with a dense nucleoid and constituted approximately 1.1% of the hepatic cytoplasm. The smaller class Q particles were also spherical and about 260 nm in diameter. They contained a granular matrix but lacked a nucleoid and their limiting membrane was trilaminar and about 130 Å in thickness. These particles constituted about 0.1-0.2% of the hepatic cytoplasm. While it is likely that the class P microbodies are peroxisomal, there is no morphological evidence to suggest that the class Q particles are small or immature peroxisomes. It is possible that the class Q microbodies are lysosomal or that they are a distinct population of hepatic cytoplasmic organelles which have not previously been described.

5379 EFFECT OF MODIFICATION OF THYROID FUNCTION ON CHOLESTEROL 7α-HYDROXYLATION IN RAT LIVER. *(E.)* Takeuchi, N.; Ito, M.; Uchida, K.; Yamamura, Y. (Osaka Univ. Hosp., Japan). *Biochem. J.* 148(3):499-503, 1975.

The effect of p.o. glucose or cholesterol administration on hepatic cholesterol synthesis and cholesterol 7α-hydroxylation was studied in hyper- and hypothyroid rats. Female Sprague-Dawley rats were fed desiccated thyroid powder (6, 18, or 54 mg/day for 10 days) to make them hyperthyroid or were fed 2-thiouracil (75 or 100 mg/day for 10 days) to make them hypothyroid. The rats were then starved and given 2 ml of 50% glucose solution/100 g and killed 3 hr later, or were given a cholesterol suspension in peanut oil (2 ml/100 g of 1.3 g cholesterol and 0.3 g cholic acid in 40 ml peanut oil) daily for 3 days. The thyroid powder and thiouracil treatments were continued during cholesterol administration. Glucose-induced increases in cholesterol synthesis and 7α-hydroxylation were enhanced by pretreatment with thyroid powder and suppressed by pretreatment with thiouracil. The enhancement of 7α-hydroxylation was produced by a relatively small amount of thyroid powder (6-18 mg), while high doses were required to increase cholesterol synthesis. In contrast, 7α-hydroxylation was suppressed by 50-75 mg thiouracil, much higher doses being required to decrease cholesterol synthesis. Although cholesterol synthesis was increased to a similar degree in both hypo- and hyperthyroid rats after glucose administration, hydroxylase activity in the hypothyroid rats increased more slowly and attained a lower maximum level than in the hyperthyroid rats. It is concluded that cholesterol 7α-hydroxylase activity is more sensitive to thyroid function than are the activities of cholesterol-synthetic enzymes. When exogeneous cholesterol was given, the hypothyroid rats showed a larger increase in the serum cholesterol concentration than the hyperthyroid rats, and there was an inverse relation between the serum cholesterol concentrations and hepatic cholesterol 7α-hydroxylase activities.

5380 MORPHOLOGICAL ALTERATION THROUGH CHANGES IN RESPIRATORY ENZYME LEVELS IN THE LIVER MITOCHONDRIA OF JAUNDICED PATIENTS. *(E.)* Takasan, H.; Yamada, T.; Ozawa, K.; Honjo, I. (Kyoto Univ. Sch. Med., Japan). *J. Electron Microsc. (Tokyo)* 24(1):19-32, 1975.

Morphological and biochemical alterations in the liver mitochondria of 20 patients with obstructive jaundice were studied. Sectioned biopsy specimens were examined by electron microscopy and mitochondrial preparations were examined in terms of phosphorylative efficiency and oxygen utilization; the respiratory enzymes of the liver mitochondria were also assayed using spectrophotometric techniques. The patients were classified into 3 groups according to the concentrations of cytochrome $a(+a_3)$ in their liver mitochondria: in group I, the cytochrome $a(+a_3)$ concentration was $0.5\text{-}1.0 \times 10^{-10}$ M/mg mitochondrial protein; in group II, the concentration was over 1.0×10^{-10} M/mg; and in group III, the concentration was less than 0.5×10^{-10} M/mg. In 6 of the 9 group I patients, the absolute phosphorylative activities of the liver mitochondria were substantially above control values and the phosphorylative activities/U of cytochrome $a(+a_3)$ were enhanced. In 6 of the 8 group II patients, the absolute phosphorylative activities were slightly reduced below control levels and the phosphorylative activities/U of cytochrome $a(+a_3)$ were inhibited. In 4 patients in group III, both the absolute phosphorylative activities and the phosphorylative activities/U of cytochrome $a(+a_3)$ were severely inhibited. Morphologically, the mitochondria in group I were round or oval in shape with an electron-lucent matrix. In group II the mitochondria were enlarged and there were increased numbers of cristae; giant- or mega-mitochondria were frequently observed. In group III, the mitochondrial cristae were decreased in number and showed an irregular arrangement; the matrix was also decreased in density with a loss of intramitochondrial granules and swelling. The various morphological changes seen in the liver mitochondria of groups I and II, including the appearance of partition- and mega-mitochondria, appeared to represent a series of adaptive

phenomena for maintaining intracellular energy metabolism in obstructive jaundice.

5381 THE EFFECT OF PROSTAGLANDIN A_1 AND E_1 ON CANINE HEPATIC BILE FLOW. *(E.)* Kaminski, D. L.; Ruwart, M.; Willman, V. L. (St. Louis Univ. Sch. Med., Mo.). *J. Surg. Res.* 18(4):391-397, 1975.

The effects of prostaglandins A_1 and E_1 on the excretion of bile salt, secretin, and cholecystokinin-stimulated hepatic bile flow were studied in dogs with chronic biliary and gastric fistulas. The dogs received a continuous i.v. infusion of 0.15 M NaCl solution or of a sodium taurocholate solution (18 μM/min). In some cases, secretin (2 U/kg/hr) or CCK-octapeptide (4 μg/kg/hr) was infused with the bile salt. The prostaglandins (2.5 μg/kg/min) were given simultaneously through separate i.v. lines. The bile and gastric secretions were collected and measured throughout the infusion periods. PGA_1 infusion caused an increase in bile volume and in both bicarbonate concentration and output; bile salt concentration was decreased and sodium and chloride output increased. Similar effects were obtained with PGE_1 infusion, although the change in bile volume was not significant. PGA_1 administered during secretin-stimulated bile flow resulted in a significant inhibition of bile flow and sodium output and a significant increase in the bicarbonate and bile salt concentrations. PGE_1 significantly increased the bile salt output and concentration under these conditions but did not affect the volume or electrolyte composition of the bile. PGE_1 inhibited the CCK-octapeptide-stimulated gastric fistular hydrogen ion output by 54.6% and significantly decreased the bile salt concentration. PGA_1 inhibited the CCK-octapeptide-stimulated gastric acid response by 40.2% and increased the sodium output and concentration but had no significant effect on bile volume. At no time did the pH of the duodenal fluid fall below 5.0. The data indicate that PGE_1 and PGA_1 are potent inhibitors of gastric acid secretion and that they are effective inhibitors of CCK-octapeptide-stimulated hydrogen ion output and pentagastrin-stimulated hydrogen ion output.

5382 EFFECT OF EXOGENOUS CYTOCHROME C ON OXIDATIVE PHOSPHORYLATION IN RAT LIVER IN ACUTE TOXIC HEPATITIS. *(Rus.)* Slepneva, L. B.; Krivoruchko, B. I. (Res. Inst. Hematol. Blood Transf., Leningrad, USSR). *Biull. Eksp. Biol. Med.* 77(6):46-48, 1974.

The effect of parenteral administration of cytochrome C on processes of oxidative phosphorylation in the liver of rats with acute toxic hepatitis, caused by a single subcutaneous injection of CCl_4, was studied. Experimental rats (160-220 g) were divided into 3 groups. Into the rats of the first group a 0.25 ml/100 g dose of 40% peach oil solution of CCl_4 was injected and the animals were killed 1, 3, or 7 days after the injection. Rats of the second group received daily injections of 10 mg/1 kg of cytochrome C beginning from the day of the CCl_4 injection. Instead of CCl_4 and cytochrome C, the third (control) group of rats were injected with equivalent volumes of peach oil and physiological solution. Results indicated that parenteral introduction of cytochrome C during acute intoxication by CCl_4 leads to an increase in intensity of the processes of respiration and phosphorylation in homogenates of rat liver and to the increase in intensity of conjugation of these processes. It is concluded that stimulation of oxidative phosphorylation processes in tissue of rat liver during acute intoxication with CCl_4 and during introduction of cytochrome C might be connected with the penetration of cytochrome C into the cell and its direct effect on intracellular structure of the liver.

5383 EFFECT OF EXCESSIVE INGESTION OF DIFFERENT KINDS OF FATS ON LIPID COMPOSITION AND THE USE OF EXOGENOUS FATTY ACIDS IN LIPID SYNTHESIS. *(Rus.)* Markelova, V. F.; Liapkov, B. G. (Inst. Nutr. USSR Acad. Med. Sci., Moscow). *Vopr. Pitan.* (4):8-11, 1974.

The objective of the study was to elucidate the effect of prolonged feeding of rats with excessive quantities of various fats on the composition of lipids of liver and blood, and to determine under experimental conditions the degree of involvement of fatty acids in synthesis of glycerides, phospholipids, cholesterol and its ethers. The experimental male rats (140-160 g) were fed for 30 days with isocaloric rations of 60 kcal/100 g of wt. The typical ration of the control group rats consisted calorie-wise of 18% proteins, 56% carbohydrates, and 26% of lard or sunflower oil. During the course of experimentation, in order to retain isocaloric conditions, a fraction of fat in the rations of experimental rats was increased at the expense of carbohydrates. The results showed that prolonged feeding of rats with rations with excessive fat content led to the increase of lipids, glycerides and of ethers of cholesterol in the liver; it also led to intensification of synthesis of these compounds, which in turn led to a deficit of phospholipids, favoring the formation of lipodystrophy of liver.

5384 THE EFFECT OF HEPATIC ISCHEMIA ON LIVER FUNCTION AND THE RESTORATION OF LIVER MASS AFTER 70 PER CENT PARTIAL HEPATECTOMY IN THE DOG. *(E.)* Mackenzie, R. J.; Furnival, C. M.; O'Keane, M. A.; Blumgart, L. H. (Wellcome Surg. Res. Inst., Univ. Glasgow, Scotland). *Br. J. Surg.* 62(6):431-437, 1975.

The effect of normothermic ischemia of the liver prior to resection was studied in relation to post-operative liver function and restoration of liver mass. Dogs (39) had partial hepatectomies to remove 70% of the liver including the left and central portions. In 17, the branches of the portal vein and hepatic artery supplying the caudate and right lateral lobes were occluded for 30, 45, or 60 min. At the end of each period after restoration of blood flow, portal hepatectomy was performed. In 17 dogs, partial hepatectomy only was performed, and in 5 controls, laparotomy (with handling of the viscera) only was performed. Serum bilirubin, alkaline phos-

phatase, SGPT, SGOT, total protein, albumin, and BSP retention were measured daily for 1 week postoperatively, and then weekly for 6 weeks. Of the 17 given partial hepatectomy only, 2 died from technical failure, 1 from unknown causes, and 3 developed obstructive jaundice. In the remaining 11, determinations were made. Alkaline phosphatase rose significantly to a peak of 290 U/liter at 1 week. Both SGOT and SGPT showed a large increase within 48 hr (300 mu/ml and 220 mu/ml, resp.) Total serum protein paralleled serum albumin, falling to its lowest value (35 g/liter) one week postoperatively. BSP retention was measured in eight dogs and showed an abnormal rise to 25% on the second postoperative day, followed by a return to normal. Liver restoration was 91% at the time of sacrifice and regenerated tissue was histologically normal. Of the 17 given ischemia plus partial hepatectomy, 15 survived. Serum alkaline phosphatase activity was elevated immediately after operation (130 U/liter), and in some dogs it continued to rise (360 U/liter by six weeks). SGOT and SGPT rose to peaks 48 hr after surgery (190 mu/ml, and 250 mu/ml, resp.). Serum total protein fell due to a fall in serum albumin, which reached its lowest value (29 g/liter) 3 days after surgery. BSP retention increased 24% on the 2nd day. Of the dogs, 7 also showed a late rise to 12-42% retention at various times between the 3rd and 6th weeks. Liver restoration was 83%. In the controls, SGOT rose to 99 mu/ml on the first day. Serum albumin fell, reaching a minimum value (34 g/liter) after 1 week. It is concluded that ischemia of the liver remnant for 1 hr before resection is compatible with survival in the dog.

5385 EARLY, REVERSIBLE PLASMA MEMBRANE INJURY IN GALACTOSAMINE-INDUCED LIVER CELL DEATH. *(E.)* El-Mofty, S. K.; Scrutton, M. C.; Serroni, A.; Nicolini, C.; Farber, J. (Temple Univ. Sch. Med., Philadelphia, Pa.). *Am. J. Pathol.* 79(3):579-596, 1975.

The role of plasma membranes in the pathogenesis of galactosamine-induced cell death was studied in the rat. Wistar rats injected i.p. with D-galactosamine (200 or 400 mg/kg), uridine (1.2 mg/g), glucosamine (400 mg/kg), or D,L-ethionine (1 mg/g). The rats were killed, the plasma membranes isolated from the livers, and 5'-nucleotidase, protein, glucose-6-phosphate, and cytochrome C oxidase determined. Membrane proteins were electrophoresed on polyacrylamide gels, and circular dichroism measurements were made of suspensions by spectropolarimeter. Liver samples were analyzed for calcium and copper content. Rates of *in vitro* nuclear RNA and protein synthesis were determined. The higher dose of galactosamine caused a 40% reduction in specific activity of 5'-nucleotidase in plasma membranes and markedly altered the circular dichroism of the membranes, as indicated by a 2-fold increase in maximum negative ellipticity at 22 nm (from -5680 to -9050). These effects were not seen in rats given the lower dose of galactosamine. Neither glucosamine nor ethionine caused cell death; 5'-nucleotidase activity was not inhibited in glucosamine-treated rats. Uridine administered simultaneously with galactosamine prevented the alterations in both 5'-nucleotidase and circular dichroism. Administration of uridine 2-2 1/2 hr after galactosamine reversed the inhibition of 5'-nucleotidase and significantly reduced the maximum negative ellipticity. Uridine could not reverse the membrane alterations after 3 hr, but it did reverse the inhibition of RNA and protein synthesis 6 hr after injection of 400 mg/kg galactosamine. Liver calcium levels rose from 2 hr following either 200 or 400 mg/kg galactosamine. With the higher dose, the calcium content remained constant at 3-4 hr and then rose sharply to a peak of 4 times the initial values at 12 hr. At 12-24 hr the level declined to twice the initial value. With the lower dose, the calcium level declined at 3-4 hr and then rose slightly to a peak value of less than twice the initial level by 12 hr. Copper content was not affected by galactosamine. Ethionine and glucosamine did not induce a rise in calcium content. It is concluded that necrogenic doses of galactosamine produce early changes in rat liver plasma membranes and in total cell calcium ions.

5386 TOXICITY OF CHENODEOXYCHOLIC ACID IN THE NONHUMAN PRIMATE. *(E.)* Morrissey, K. P.; McSherry, C. K.; Swarm, R. L.; Nieman, W. H.; Deitrick, J. E. (New York Hosp.-Cornell Med. Cent., New York, N.Y.). *Surgery* 77(6):851-860, 1975.

The toxicity of chenodeoxycholic acid was studied in the baboon. Pregnant and nonpregnant female baboons (*Papio anubis* and *P. cyanocephalus*) had the gallbladder sutured to the anterior abdominal wall during exploratory laparotomy, and were assigned to control (8) or chenodeoxycholic acid-treated groups (10). The treated group was given 18 mg/kg/day chenodeoxycholic acid in 0.5 cc ethanol in an orange, while controls were given oranges with ethanol only. Over a 6-month period the treated group was divided into a low-dose group of 2 baboons given 20 mg/kg/day for 14 months; an incremental-dose group of 10 which were progressed by 2 mg/week from 18 to 38 mg/kg/day over 2 1/2 months in a total experimental period of 15 months; and a high-dose group of 6 given 38 mg/kg/day for 8 months. Animals were observed daily for well-being, behavior, menstruation, food consumption, and character of food droppings or excreta. Weight and clinical chemistry were determined monthly. Percutaneous liver biopsies and gallbladder bile aspirates were obtained periodically. Bile pool salt size was determined in 1/2 of the control and experimental groups by isotope dilution kinetic techniques. There were no gross clinical changes in the experimental group throughout the study period. Percentages of chenodeoxycholic and lithocholic acids in the bile increased significantly in all treated animals. Those in the high-dose group showed maximum increases to over 80% chenodeoxycholic and 9% lithocholic acids with 1 month of treatment. Those in the low-dose and incremental-dose groups required 2-10 months to develop similar changes. Cholic and deoxycholic pools decreased significantly after 1 yr. Treated animals showed an increase in chenodeoxycholic acid pools of 181-943 mg and a decrease in the cholic acid pool of from 302-114 mg. Serum bilirubin, total

protein with albumin-globulin ratio, calcium, and phosphorus remained normal. Serum cholesterol varied as a function of pregnancy in all animals. Treatment had no effect on serum cholesterol or cholesterol pool size. The SGPT, SGOT, 5'-nucleotidase, and alkaline phosphatase tests were good indicators of response to treatment. Of the 18 treated animals, 15 developed SGOT or SGPT levels over 75 IU and moderate to severe lesions. The low- and increment-dose animals took 5-11 months to reach this point, while the high-dose group took 1 1/2-3 months. In 9 the changes occurred synchronously and were accompanied by elevations of 5'-nucleotidase and alkaline phosphatase. When both enzymatic and histologic abnormalities had appeared in the 11, the percentage of chenodeoxycholic acid has risen to over 80% in 8, 60% in 2 and 45% in 1, and lithocholic acid had risen to 8-14%. It is concluded that chenodeoxycholic acid is hepatotoxic to baboons at 20-38 mg/kg/day.

5387 25-HYDROXYVITAMIN D_3: EVIDENCE OF AN ENTEROHEPATIC CIRCULATION IN MAN. *(E.)*
Arnaud, S. B.; Goldsmith, R. S.; Lambert, P. W.; Go, V. L. W. (Mayo Clin., Rochester, Minn.). *Proc. Soc. Exp. Biol. Med.* 149(2):570-572, 1975.

The intestinal and plasma distribution of i.v. administered 25-hydroxyvitamin D_3 was studied. Normal adults (3) were intubated with a triple-lumen nasoduodenal tube after an overnight fast. The first aperture was proximal to the ampulla of Vater, the second at the ligament of Treitz, and the third 30 cm distal to the second. Polyethylene glycol (5 g/liter) was perfused at 2 ml/min through the first lumen, and duodenal aspirates collected through the second for 30-36 hr. Samples were taken and reinfused through the third. Chromium sesquioxide (0.5 g daily) was started p.o. just prior to an i.v. bolus injection of 8-10 μCi of 26, 27-^{3}H-25-OH-D_3. Serial blood specimens and all urine and feces excreted for 12 days after injection were collected. Total ^{3}H in stool samples and duodenal aspirates were determined by liquid scintillation counting. Fecal data were corrected, on the basis of the chromium sesquioxide marker, to correspond to the appropriate blood and duodenal samples. To determine disappearance of ^{3}H-25-OH-D_3 from the blood, serum was extracted with methanol-chloroform, and chromatographed. Within 5 min of the injection of the labeled vitamin, 75-86% of the ^{3}H could be found in the plasma. During the first 6 hr, 45% of the dose disappeared from the plasma and 17.1% of the dose appeared in the duodenum. Plasma radioactivity dropped to 26.7% of the dose at 24 hr, while cumulative intestinal radioactivity doubled to 35.5%. Fecal radioactivity corresponding to the first 24.7 hr was 3.03% of the dose, indicating resorption of at least 85% in the duodenum. Total fecal excretion of ^{3}H (22.5% in 8.9 days) was much lower than the quantity of ^{3}H in the duodenum in the first 24 hr. Urinary excretion accounted for 1.47% after 24 hr, and 2.30% after 48 hr. The intestinal reabsorption of 25-hydroxyvitamin D_3 is indicative of an enterohepatic circulation that may be of physiologic importance. Interruption in the recycling of this vitamin D_3 metabolite may be an important mechanism of acquired vitamin D deficiency in gastrointestinal disease.

5388 DEUTERIUM TRANSFER FROM [1,1-^{2}H]ETHANOL DURING METABOLISM OF BILE ACIDS AND CYCLOHEXANONE IN THE ISOLATED PERFUSED RAT LIVER. *(E.)*
Cronholm, T.; Erliksson, H.; Matern, S.; Sjovall, J. (Kemiska Inst. Stockholm, Sweden). *Eur. J. Biochem.* 53(2):405-410, 1975.

Factors in the transfer of deuterium from (1,1-^{2}H)-ethanol to reduction products of cyclohexanone, 3-oxo-5β-cholanoic, and 3-oxo-4-cholenoic acids were studied in the isolated, perfused rat liver. Livers from Sprague-Dawley rats were perfused with 3-oxo-5β-cholanoic acid (males), or 3-oxo-4-cholenoic acid (females). The perfusate contained 12 ml blood diluted to 72 ml with Tyrode's solution, with a ^{2}H excess of 46 atoms% as ^2H_2O. Unlabeled or (1,1-^{2}H)-ethanol was added to give a concentration of 69 mM, and 1 mg 3-oxo-4-cholenoic acid or 1 mg 3-oxo-5β-(24-^{14}C) cholanoic acid (10^6 cpm) was added. Bile was collected in 1 hr portions, and lithocholic acid, 3-hydroxy-5β-cholanoic acid, and cyclohexyl glucuronide were extracted. With 3-oxo-5β-cholanoic acid as the substrate, lithocholic and chenodeoxycholic acids were usually found as metabolites, the former constituting 10-80% of the metabolites. No bile acids containing a 3-oxo group were found. Lithocholic acid formed from 3-oxo-5-β during (1,1-^{2}H)-ethanol perfusions contained 11% monodeuterated molecules, and none had more than one deuterium atom. Abundance of deuterated molecules was diminished by cycloserine or aminooxyacetate in the perfusion medium, and maleate, disulfiram, lactate, and malate caused a decrease in labeling. Abundance of monodeuterated cyclohexanol molecules was 49% during the metabolism of the labeled ethanol. Deuterium excess in cyclohexanol was increased by maleate, lactate, malate, and oleate. Abundance of monodeuterated lithocholic acid molecules formed in ^2H_2O medium (46 atoms % excess) was 26%, and an insignificant number had more than one deuterium atom. When the acid was oxidized and the product converted to *O*-methyloxime, abundance of monodeuterated molecules decreased to 2.8%, showing most of the deuterium in the acid was in the 3β position. There was no substantial loss of deuterium in the conversion of (2,2,4,4 ^{2}H)lithocholic acid into the *O*-methyloxime of the 3-oxo derivative. Unlabeled ethanol decreased abundance of monodeuterated lithocholic acid molecules to 21%; when (1,1-^{2}H)ethanol was present the abundance increased to 29%. Excreted 3α-hydroxy-5-cholanoate sulfate had a deuterium excess in 20 atoms % in the 3β position and of 24 atoms % in the C-2 and C-4 positions. It is concluded that different pools of NADPH are used for reduction of the 4,5 double bond and of the 3-oxo group.

5389 TWO DISTINCT PATTERNS OF GLUCOSE INTOLERANCE IN ICTERIC RATS AND RABBITS. RELATIONSHIP TO IMPAIRED LIVER MITOCHONDRIA FUNCTION. *(E.)* Yamada, T.; Ida, T.; Yamaoka, Y.; Ozawa, K.; Takasan, H.; Honjo, I. (Kyoto Univ. Fac. Med., Japan). *J. Lab. Clin. Med.* 86(1):38-45, 1975.

The correlation between the degree of glucose tolerance and the degree of liver mitochondrial derangement was studied in animals with liver disease. Rabbits and Wistar rats had the common bile duct doubly ligated close to the liver, and the left branch of the portal vein ligated. P.o. glucose tolerance test was performed after a 15-hr fast in rats subjected to ligation for 3 weeks and rabbits subjected to ligation for 48 hr. Glucose (3 g/kg in rats and 2 g/kg in rabbits) in 20% solution was given intragastrically, and blood samples were taken at intervals up to 6 hr. Insulin tolerance was determined by i.v. injection of insulin (1 U/kg in rats, and 0.2 U/kg in rabbits). Liver mitochondria were assayed for oxygen consumption, phosphorylative efficiency, respiratory carrier content, protein, and respiratory control ratio. In icteric rats, blood glucose peak was reached in 2 hr, in contrast to 1 hr for controls, and was significantly higher (159 mg/dl) than in controls (137 mg/dl). The plasma immunoreactive insulin peak was reached in 1 hr, in contrast to 30 min for controls, and was significantly higher than in controls. In icteric rabbits, blood glucose level increased gradually for 6 hr to a significantly higher level. Plasma immunoreactive insulin response peaked at 3 hr, compared with 30 min for controls, and was significantly higher than in controls. In icteric rats, blood glucose and plasma immunoreactive insulin levels were not significantly different from controls. In icteric rabbits, blood glucose levels at 15 and 30 min after insulin injection were significantly higher than those in controls. In both rats and rabbits with ligated portal veins, phosphorylase/unit of cytochrome a ($+a_3$) decreased rapidly to 15/sec in 24 hr and remained constant. In rats with bile duct ligation, the activity per unit of a($+a_3$) cytochrome fell to 20/sec in 2 weeks and remained constant. In the icteric rabbits, activity rapidly decreased to 10/sec in 24 hr after ligation. Respiratory control ratio, State 3 respiration, and phosphorylase activity/mg protein/min were significantly higher in icteric rats than those with ligated portal veins. Of the icteric rats, 90% lived beyond three weeks, while nearly all the icteric rabbits died within three days. It is concluded that glucose intolerance in icteric rats and rabbits is related to severity of liver mitochondrial derangement.

5390 SPECIFIC DEUTERIUM LABELLING AND COMPUTERIZED GAS CHROMATOGRAPHY-MASS SPECTROMETRY IN STUDIES ON THE METABOLISM IN VIVO OF A STEROID SULPHATE IN THE RAT. *(E.)* Baillie, T. A.; Eriksson, H.; Herz, J. E.; Sjovall, J. (Dep. Chem., Karolinska Inst., Stockholm, Sweden). *Eur. J. Biochem.* 55(1): 157-165, 1975.

Specific deuterium labeling and computerized gas chromatography-mass spectrometry were used to study the metabolism of allopregnan-3β-01-20-one sulfate in bile fistula rats and in isolated perfused rat livers. The deuterium-labeled steroid (about 0.5 mg) was dissolved in 50% ethanol and isotonic glucose solution and injected i.v. into male and female Sprague-Dawley rats with bile fistulas. The steroid, again dissolved in ethanol and isotonic glucose, was added to the perfusate 20-100 min after the start of perfusion of isolated livers. The biliary steroids in each case were extracted from the bile samples collected during steroid treatment, purified, and analyzed by gas-liquid chromatography and combined gas chromatography-mass spectrometry. The male rats excreted a-lopregnan-3β-01-20-one sulfate metabolites into the bile more rapidly than did the females, an observation which was correlated with the preferential formation of glucuronide conjugates in the male liver. The major metabolic pathway in the males involved hydrolysis, 2α-hydroxylation, oxidoreduction at C-3, and glucuronide conjugation, to yield 2α,3α-dihydroxy-5α-pregnan-20-one glucuronide as the major metabolite; only traces of the injected steroid sulfate were detected in bile from male rats. In contrast, the injected steroid sulfate was the major steroid excreted in the bile of the female rats; in this case, the primary metabolite was identified as 3β,15β-dihydroxy-5α-pregnan-20-one sulfate. A minor metabolite, 3β,16α-dihydroxy-5α-pregnan-20-one, was found as a monosulfate in the females and as both a disulfate and a glucuronide conjugate in the males. The deuterium content of the sulfated 15β- and 16α-hydroxylated metabolites was consistent with metabolic pathways involving direct hydroxylation of the injected steroid sulfate. Similar results were obtained in the liver perfusion experiments, which indicates that all the observed metabolic reactions took place in the liver. The results demonstrate the advantages associated with the use of stable isotopes in studies of steroid metabolism.

5391 SERUM CALCIUM AND HEPATIC KUPFFER CELL PHAGOCYTOSIS. *(E.)* Ryder, Jr., K. W.; Kaplan, J. E.; Saba, T. M. (Albany Med. Coll., N.Y.). *Proc. Soc. Exp. Biol. Med.* 149(1):163-167, 1975.

The effect of experimental alterations of serum calcium levels on opsonin-mediated hepatic phagocytosis was investigated in male Holtzman rats. Serum calcium concentration was altered by the addition of calcium chelators (oxalate, citrate, and EDTA) or by the addition of exogenous calcium. The test colloid used for the phagocytic studies was gelatinized "reticuloendothelial test lipid emulsion"; hepatic phagocytosis was assayed *in vitro* using an isotopic liver slice technique. Upon the addition of oxalate, phagocytosis was depressed in proportion to the amount of oxalate added. At the highest concentration of the chelator (2 μM/ml serum), phagocytosis was 49.6% of control with oxalate, 35.8% of control with citrate, and 48.3% of control with edetic acid. When exogenous calcium was added to serum pretreated with oxalate or citrate, enhancement of phagocytosis occurred with calcium levels up to normal; at calcium levels above normal, there was a significant depressant effect on phagocytosis. The findings suggest that alterations of serum calcium may induce significant changes in reticuloendothelial function which may produce a state of altered host resistance.

5392 LIVER REGENERATION: AN ISOLATION PERFUSION SYSTEM EMPLOYED TO ASSAY HEPATIC ^{3}H-THYMIDINE INCORPORATION. *(E.)* Sera, Y.; McLeod, J. W.; Koga, Y.; Nicolson, M. O.; Hays, D. M. (Child. Hosp. Los Angeles, Calif.). *Proc. Soc. Exp. Biol. Med.* 149(1):294-298, 1975.

The ability of hepatic cells to incorporate a labeled DNA precursor was studied using an isolation-perfusion apparatus. Male Sprague-Dawley rats were subjected to either a 68% hepatic resection or sham-hepatic mobilization. Isolated, whole normal rat livers were perfused with 2 types of suspension, perfusate that had passed through a regenerating liver and perfusate that had passed through a "sham" operated liver. After the initial 10 min of perfusion, 125 μCi thymidine-me-^{3}H was introduced into the perfusate, and perfusion was continued for 60 min. The livers were then removed and small pieces were fixed for autoradiographic studies; DNA was extracted from the remaining liver. Isolated normal livers perfused with suspension which had been previously circulated through either regenerating or sham-operated livers showed equal and relatively low levels of both tissue specific activity and nuclear labeling. When fresh perfusates were circulated through regenerating livers, nuclear uptake was apparent and specific activity was increased more than 100 times that found in similarly perfused sham-operated livers. No evidence of a factor which can rapidly increase the ^{3}H-thymidine uptake in a perfused normal liver was detected in these studies. The hepatic perfusion system represents a method of demonstrating the capacity for active DNA synthesis in liver cells, employing perfusion as a substitute for i.v. or i.p. injection of labeled compounds in intact animals.

5393 EFFECT OF INTRA-ARTERIAL INFUSION OF VASOPRESSIN ON HEPATIC ARTERY FLOW. *(E.)* Fingeroth, R. J.; Storer, E. H.; Ramsby, G. R.; Conn, H. O. (VA Hosp., West Haven, Conn.). *Surg. Forum* 24:399-402, 1973.

The possibility of hepatic ischemia developing secondary to decreased hepatic artery blood flow after vasopressin administration was studied in 15 dogs. Noncannulating electromagnetic flow probes were used to monitor blood flow through the hepatic and splenic arteries and portal veins following the intraceliac infusion of 10 U of vasopressin. The preinfusion and postinfusion angiograms of ten patients undergoing therapeutic intraceliac vasopressin infusion were also studied. Upon the arrival of vasopressin in the celiac axis, hepatic artery flow fell to 65% of baseline values, and splenic artery flow fell to 50% of baseline values. Over the next min, the systemic blood pressure rose to 134% of baseline values and the portal vein flow fell to 51% of baseline values. As the portal vein flow decreased, the hepatic artery flow increased, reaching a mean value of 136% of baseline values. This state remained constant throughout vasopressin infusion, with all flows returning to baseline values postinfusion. In the ten human patients, the postvasopressin infusion angiograms showed no change, or showed an actual increase in hepatic artery diameters, while the splenic, gastric, and gastroduodenal arteries showed marked distal vasoconstriction. It is concluded that hepatic artery infusion of vasopressin is not hazardous provided compensatory dilatation occurs as documented by angiography after a brief trial infusion.

5394 COPPER METABOLISM AFTER BILIARY FISTULA, OBSTRUCTION, OR SHAM OPERATION IN RATS. *(E.)* Owen, C. A., Jr. (Dep. Biochem., Mayo Clin., Rochester, Minn.). *Mayo Clin. Proc.* 50(7):412-415, 1975.

The excretion of copper was studied after biliary obstruction in male Sprague-Dawley rats. The animals were subjected to surgical procedures whereby the bile duct was cannulated and the cannula was threaded out through the skin, the bile duct was doubly ligated and the intervening segment resected, or laparotomy and exposure of the bile duct was followed by simple resuturing of the wound (sham operation). All rats were given vitamin K (0.5 mg) s.c. every other day and i.v. ^{67}Cu (1 μCi) injections. The carrier-free ^{67}Cu appeared rapidly in the bile of the rats with recently-created bile fistulas, maximum excretion occuring within the first 2 hr. However, in animals in which the biliary fistula had been created 3-4 days previously, only one to one-half as much of the isotope appeared in the bile. There was also a small decrease in stable biliary copper in these rats. When the biliary fistula rats were compared over a period of 6 weeks to those with permanent biliary obstructions, both groups exhibited a marked increase in plasma copper and an equally pronounced increase in plasma *p*-phenylenediamine oxidase activity; there was no increase in hepatic copper. Peaks in the blood copper levels were reached within 2 weeks and then slowly diminished. In the sham-operated rats, the copper and enzyme changes paralleled those in the other groups but were much less marked. The results suggest that in inherited copper retention (Wilson's disease) or acquired copper retention (primary biliary cirrhosis), there may be a pathogenic abnormality in the bile which induces copper retention in these diseases. The results also suggest that the absence of bile in the gut alters copper metabolism.

5395 ULTRASTRUCTURAL CHANGES IN RAT HEPATOCYTES FOLLOWING ACUTE METHYL MERCURY INTOXICATION. *(E.)* Desnoyers, P. A.; Chang, L. W. (Dep. Pathol., Univ. Wisconsin, Madison). *Environ. Res.* 9(3):224-239, 1975.

The pathological and morphological effects of acute and chronic methyl mercury intoxication on the rat liver were studied. Male Sprague-Dawley rats were given up to four consecutive daily s.c. injections of methylmercuric chloride at a dosage of 10 mg/kg. The animals were fasted for 24 hr before sacrifice and were killed 1, 5, 10, 24, 48, or 96 hr after the beginning of methyl mercury treatment. Ultrathin sections of the livers were examined by electron microscopy. No neurological symptoms were observed in any of the animals. Grossly, the livers appeared atrophied and adhesion of the liver to the visceral

organs occured after the second injection. Ultrastructural changes in the hepatocytes were observed as early as 5 hr after the initial mercury injection; these included a wavy appearance of the mitochondrial membrane, dilatation of the rough endoplasmic reticulum, and occasional accumulation of electron-dense granules (liposomes) near the bile canaliculi. After 24 hr, there was a general depletion of glycogen and focal amorphous areas appeared in the cytoplasm of many cells. Large areas of cytoplasmic degradation were also observed and cellular materials were frequently observed to be extruded into the hepatic sinusoids. Extensive matrical swelling of the mitochondria with concomitant loss of the mitochondrial granules were observed after 2 days; breakage of the outer mitochondrial limiting membranes, loss of the mitochondrial cristae, and an increase in the number of lysosomes were also noted at this time. Sequestration of cytoplasmic organelles, many of them degraded, within the hepatocytes to form large membrane-limited cytosegresomes was observed after 4 days. Degenerated organelles, Kupffer cells and cellular debris were frequently found extruded into the hepatic sinusoids and moderate fatty changes in some hepatocytes were observed. The data indicate that the most extensive degenerative changes in the liver occur after 2 days of methylmercury administration.

5396 TECHNETIUM ^{99m}-PYRIDOXYLIDENEGLUTAMATE: A NEW HEPATOBILIARY RADIOPHARMACEUTICAL. I. EXPERIMENTAL ASPECTS. *(E.)* Baker, R. J.; Bellen, J. C.; Ronai, P. M. (Inst. Med. Vet. Sci., Adelaide, South Australia). *J. Nucl. Med.* 16(8):720-727, 1975.

The electrophoretic behavior, *in vivo* distribution, toxicity, and reabsorption of technetium-99m-pyridoxylideneglutamate (^{99m}Tc-PG) were studied in balb/c mice and female rats to determine the potential of this radiopharmaceutical for the scintigraphic diagnosis of biliary tract disorders in man. Labeling of pyridoxal and the pyridoxylidene derivative of glutamic acid with ^{99m}Tc was achieved by a simple autoclaving procedure, after which the autoclaved reaction mixture was examined using electrophoresis on cellulose acetate membrane. The compound (75 to 500 μCi) was injected i.v. into balb/c mice and dogs, and its biliary and urinary excretion and tissue distribution were studied; the acute toxicity of PG was also determined in mice, and its antigenicity was studied in guinea pigs. In the reabsorption experiments, radioactive bile from a donor rat given i.v. ^{99m}Tc-PG was injected into the duodenum of a recipient rat and the radioactivity in the blood, bladder, bile and urine was determined at 15-min intervals. Both scintigraphic and quantitative distribution studies showed that ^{99m}Tc-PG passed rapidly through the mouse liver with progressive accumulation in the gallbladder, allowing visualization of this organ within 10 min of injection. Within 30 min, over 40% of the injected dose was excreted into the intestine with an equivalent amount appearing in the urine; renal activity was low. Scintigraphic studies in dogs yielded similar results. The LD_{50} of this compound in mice was about 1,400 mg/kg, but there were no apparent toxic reactions or histological changes noted after the cumulative administration of 2,820 mg/kg in ten equal doses. The preparation was sterile, pyrogen-free, and did not produce an antigen reaction in guinea pigs. In the reabsorption experiments, the radioactivity appearing in the blood of the recipient at 2 hr reached 0.02% of the dose/ml; no activity was observed in the bladder at this time. It is concluded that ^{99m}Tc-PG would be a suitable agent of low toxicity for use in humans for the investigation of biliary tract disorders.

5397 ENDOTOXIN-INDUCED LIVER NECROSIS AND INTRAVASCULAR COAGULATION IN RATS ENHANCED BY PORTACAVAL COLLATERAL CIRCULATION. *(E.)* Liehr, H.; Grun, M.; Thiel, H.; Burnswig, D.; Rasenack, U. (Dep. Med., Univ. Wurzburg, Germany). *Gut* 16(6):429-436, 1975.

The effects of i.v. endotoxin on the hepatic and systemic circulation and on the coagulation system were evaluated in normal, non-fasted FW 49 rats and in rats with experimental portal hypertension or portacaval anastomosis. Endotoxin (1.5 mg/kg) was injected into the penile vein and the cardiac output and hepatic blood flow were measured. Coagulation factors II, V, VII, and X were also determined, as were the fibrinogen level and platelet count, the activities of SGOT and SGPT, phagocytic activity, and the serum bilirubin and blood sugar concentrations. Specimens of liver, lungs and kidney were examined histologically. In the normal rats, endotoxin administration led to a prompt increase in the serum transaminase activity and to a hyperdynamic circulation with a consequent increase in the total hepatic blood flow. Six hr later, the hepatic artery dilated with a subsequent hepatic arterial hyperperfusion. The coagulation system was also affected, with signs of consumption coagulopathy. Endotoxin treatment resulted in acute liver necrosis within 12-15 hr in the rats with portal hypertension and portacaval collaterals as well as in those with portacaval anastomosis. The hepatic artery became overdilated with a cardiac output fraction of 25%, compared to a normal value of 5.5%. Blood extravasates and thrombi, rich in fibrin, were detected in the liver. Hypocoagulation, greatly elevated transaminase activity, and greatly reduced blood sugar levels were also observed. It is suggested that the exaggerated endotoxin effect in these rats was due to an impairment in the clearance function of the reticuloendothelial system, probably as a consequence of portacaval collateral circulation. It is concluded that endotoxins damage the liver, even in normal organisms, that they are potent inducers of acute liver necrosis if the reticuloendothelial system is altered, and that they should be considered in the pathogenesis of acute and chronic liver diseases.

5398 MORPHOLOGICAL FINDINGS IN SWINE LIVER HOMOGRAFTS AFTER ORTHOTOPIC AND AUXILIARY HETEROTOPIC TRANSPLANTATION. *(Ger.)* Seidler, D.; Trautwein, G.; Lauchart, W.; Bockhorn, H. (Veterinary Med. Coll., Hannover, Germany). *Z. Immunitaetsforsch.* 148(1):1-15, 1974.

Morphological and immunohistochemical studies were performed on the livers of donor and recipient swine before and after transplantation and after the death of the recipient, 2 days to 1.5 yr later. Orthotopic transplants were performed on 20 swine and heterotopic auxiliary transplants on ten. No immunosuppressive agents were employed. Gross examination of the livers of swine that died within 5 days after surgery revealed hepatomegaly and changes in blood supply. One animal had a completely necrotic left lobe with no signs of a thrombus in branches of the hepatic artery or portal vein supplying this area. In swine surviving 5 days to 3 weeks, boundaries between the lobes and the periportal fields were more clearly defined than in normal animals; almost half of the swine had hepatomegaly. Rejection was manifested by parenchymal bleeding and small areas of necrosis which gave cut sections a variegated appearance. Of the swine receiving orthotopic transplants, 2 developed total and 2 others partial stenosis of the common bile duct near the suture line and intrahepatic cholestasis. In swine surviving longer than 3 weeks, periportal fields became wider, the hepatic capsule was thickened and fibrotic, and lobular architecture was lost. Nonspecific histological changes found immediately after surgery included marked dilatation of central veins and sinusoids, centrilobular hepatocyte dissociation, and Kupffer cell activation. Large centrilobular vacuoles, nuclear degeneration, isolated cellular necrosis, and edema of periportal tissue were observed in swine that died within 5 days after surgery. In animals that survived longer, lymphocyte infiltrations, isolated cellular necrosis, and fibrotic widening of the boundaries between lobules were detected. Other nonspecific changes included leukocyte infiltration of the bile ducts and bile duct proliferation which developed in association with stenosis and partial stenosis of the common bile duct. Morphological changes resulting from immunological differences between the donor and recipient varied with the survival time and the intensity of the immune reaction; no appreciable differences were found between orthotopic and auxiliary heterotopic transplants. These changes consisted of lymphocyte infiltration beginning in the portal triads and eventually resulting in fibrosis. The fluorescent antibody technique failed to demonstrate that humoral antibodies participate in the rejection reaction.

5399 IS GLUCOSE THE PORTAL HEPATOTROPHIC SUBSTANCE? *(E.)* Mooney, C. S.; Griffen, W. O., Jr. (Univ. Kentucky Med. Cent., Lexington). *Surg. Forum* 24:379-381, 1973.

The possible role of glucose in preventing liver shrinkage following substitution of systemic for portal venous blood was studied in adult dogs. Partial portacaval transposition was performed in each animal so that the right hepatic lobe received splanchnic venous blood while the left lobe received systemic venous blood. For five days following surgery, the dogs were given commercially available horse meat once daily and water *ad libitum*. For five hr after eating, half of the dogs received 500 ml of saline at a rate of 100 ml/hr i.v. in a hind limb; the others received a similar amount of 5% dextrose in saline. There were no significant alterations in the SGOT, bilirubin, or alkaline phosphatase values after surgery in any of the animals, and all maintained their appetite and weight throughout the study. At 60 days after surgery, the right liver lobes appeared normal in all dogs and the left lobes appeared normal in the glucose group. The left lobes in the saline group were paler, significantly smaller, and firmer than normal, weighing on an average only 249 g compared to 410 g in the glucose group. Histologically, the portions of liver receiving splanchnic blood were normal; the left lobes in the glucose group showed some mononuclear infiltration at the portal triads, and the left lobes of the saline group showed increased fibrous tissue and small, irregular, darker hepatocytes. The results support the hypothesis that depriving the liver of portal venous inflow leads to a "starvation" sequence; that portion of the liver which does not receive postprandial glucose-laden blood cannot metabolize and store glycogen and therefore undergoes shrinkage. The role of insulin in this process appears to be less critical than exposure of the hepatocytes to glucose.

5400 LIVER, BILIARY SYSTEM, AND GASTROINTESTINAL MUCOSA: SITE OF ORIGIN OF THE HEPATOTROPHIC PORTAL BLOOD FACTOR INVOLVED IN LIVER REGENERATION. *(E.)* Sgro, J. C.; Charters, A. C.; Chandler, J. G.; Grambort, D. E.; Orloff, M. J. (Univ. California, San Diego Sch. Med., La Jolla). *Surg. Forum* 24:377-379, 1973.

Liver regeneration was examined in male Lewis rats after selective ablation of the splanchnic organs to study the site of origin of the hepatotrophic portal blood factor responsible for liver regeneration. All animals were starved for 48 hr then subjected to 70% hepatectomy. In addition, some were subjected to small-intestine resection; resection of the spleen, stomach, duodenum, and pancreas; resection of the stomach, spleen, and duodenum; pancreatectomy; or gastrectomy. Biliary and gastrointestinal continuity were reestablished by anastomoses. All animals were killed 24 hr after surgery, having been injected i.p. with tritiated thymidine (250 μc) 30 min prior to sacrifice. The mitotic index, labeled nuclei, and DNA uptake of ^{3}H-thymidine were examined in the remnant liver. Marked regenerative activity of the residual liver segment was evident in the animals which had undergone hepatectomy, hepatectomy plus small-intestine resection, and hepatectomy plus gastrectomy, the magnitude of the response being reduced in the latter 2 groups compared with the first group. No regenerative activity was noted after hepatectomy plus removal of the stomach, spleen, pancreas, and duodenum, or after hepatectomy plus removal of the stomach and duodenum or the pancreas alone. The data indicate that the hepatotrophic portal blood factor is not a nutrient substance absorbed from the gastrointestinal tract. They also suggest that the site of origin of this factor is not the small intestine or stomach, although the pancreas and/or duodenum may prove to be the source of this humoral factor.

5401 ANIMAL MODEL OF HUMAN DISEASE: CHRONIC BILIARY OBSTRUCTION. *(E.)* Stanley, N. F.; Joske, R. A. (Dep. Microbiol., Univ. West. Australia, Nedlands). *Am. J. Pathol.* 80(1):185-187, 1975.

A murine model is described which might be used to study the clinical and pathologic features of chronic biliary obstruction in man. Nonbacterial infections producing chronic biliary obstruction in humans are at present an enigma. Obstruction may result from a viral hepatitis-induced cholestatic syndrome, fibrous strictures at the lower end of the common bile duct in the absence of either pancreatitis or cholelithiasis, or from viral infections which produce biliary obstructions secondary to viral pancreatitis. The i.p. inoculation of weanling mice with massive doses of reovirus Type 3 resulted in a 40% incidence of chronic obstructive jaundice within 3 or more weeks. Jaundice resulted from the obstruction of the common bile duct at the duodenal end following denudation of the bile duct mucosa. One week after inoculation, reovirus was observed replicating in the bile duct mucosal cells, but no infective virus could be detected at 3 weeks. Virions passed both the sinusoidal and hepatic layers before reaching the common bile duct, the cells lining its lumen being invaded when the viral titer in the duct became high enough. The cellular necrosis and inflammatory response to virus invasion were followed by fibrosis in the terminal portion of the common bile duct. The subsequent stricture formation and obstruction of flow in the common bile duct were responsible for the hepatic and pancreatic lesions seen in the later stages of the disease. This model suggest that some cases of biliary obstruction at the ampulla of Vater might occur following viral choledochitis. A combined clinical and epidemiologic approach with attempted virus isolation from blood and feces, as well as viral and electron microscopic study of possible surgical specimens, might aid in the study of chronic biliary obstruction in man.

5402 RAPID UPTAKE BY LIVER SINUSOIDAL CELLS OF SERUM ALBUMIN MODIFIED WITH RETENTION OF ITS COMPACT CONFORMATION. *(E.)* Buys, C. H. C. M.; De Jong, A. S. H.; Bouma, J. M. W.; Gruber, M. (Biochem. Lab., Univ. Groningen, The Netherlands). *Biochim. Biophys. Acta* 392(1):95-100, 1975.

The conformation and blood clearance of nitroguanidinated albumin were studied using nitroguanidinated bovine serum albumin and native albumin, labeled with ^{125}I and injected into rats. The radioactivity in the plasma and liver were determined in the first hours after injection, and the subcellular localization of the protein was compared with that of the lysosomal enzymes acid phosphatase and cathepsin D. The nitroguanidination-induced changes in the albumin molecule were studied by gel filtration on Sephadex G-200 and by chemical characterization of native, nitroguanidinated, and formaldehyde-treated albumin. Nitroguanidination resulted in replacement of the positively-charged amino groups on the albumin molecule by neutral nitroguanido groups. However, there was no significant folding of the modified albumin molecule and the number of reducible disulfide groups was not appreciably increased. Like formaldehyde-denatured albumin, but in contrast to the native protein, the nitroguanidinated derivative was rapidly cleared from the blood *in vivo* and was taken up in the lysosomes of the liver sinusoidal cells. It is concluded that extensive denaturation is no prerequisite for the uptake of albumin derivatives by the liver sinusoidal cells. It is further suggested that the nitroguanidinated protein, in contrast to native albumin, is bound on membrane receptors of these cells. The nitroguanidine groups themselves may be bound on these receptors, but it seems equally possible that binding on the receptors is the result of a net increase in the negative charge of the albumin molecule or of local conformational changes in the protein.

5403 EFFECT OF DECREASED GALLBLADDER STIMULATION ON ENTEROHEPATIC CYCLING AND KINETICS OF BILE ACIDS. *(E.)* Hepner, G. W. (Milton S. Hershey Med. Cent., Pennsylvania State Univ., Hershey). *Gastroenterology* 68(6):1574-1581, 1975.

The effect of dietary protein and fat on the rate of enterohepatic recycling and bile acid kinetics was studied in patients with cholesterol cholelithiasis, in cholecystectomized patients and in control subjects. The effect on enterohepatic cycling was assessed indirectly by comparing the 24-hr output of breath $^{14}CO_2$ after administration of 2 μCi of cholyl (1-^{14}C)glycine during two periods: a control period when three meals containing protein and fat were eaten daily; and a diet period, when protein and fat were excluded from the diet for a period of 72 hr. Bile acid kinetics after the p.o. administration of (2,4-^{3}H)cholic acid and (24-^{14}C)chenodeoxycholic acid were measured in six healthy subjects during a control period and again, not less than 4 months later during the fourth week of a dietary program in which one meal containing fat and protein was consumed every other day. In the first study, breath $^{14}CO_2$ output was not altered by diet in the cholecystectomy patients. In the healthy subjects and in the patients with cholesterol cholelithiasis, however, the breath $^{14}CO_2$ output fell by about 50%; this indicated that decreased endogenous cholecystokinin-pancreozymin stimulation had reduced but not eliminated enterohepatic recycling in these subjects. In the second study, the pool size of cholic and chenodeoxycholic acid in the bile rose significantly during the low fat and protein diet; the total bile acid pool size also decreased. The daily fractional turnover rate of both primary bile acids fell significantly during the diet, but their rate of synthesis was not significantly changed. It is concluded that significant enterohepatic circulation of bile acids occurs even in the absence of dietary stimuli for gallbladder contraction and that diet may significantly affect bile acid pool size and fractional turnover, while bile acid synthesis remains essentially unaffected.

5404 HEPATIC BLOOD FLOW AND OXYGEN CONSUMPTION IN STARVATION, SEPSIS AND SEPTIC SHOCK. *(E.)* Imamura, M.; Clowes, G. H. A., Jr. (Harvard Med. Sch., Boston, Mass.). *Surg. Gynecol. Obstet.* 141(1):27-34, 1975.

The normal pattern of hepatic circulation and oxygen consumption in the fasted state was compared with that observed during sepsis. Sepsis was induced in male and female pigs by cecal ligation, which produced a fulminating i.p. abscess containing coliform organisms. Prior to the induction of sepsis, the pigs were fasted for 4 days, thermodilution catheters being inserted into the portal vein and inferior vena cava on the third day; silastic tubes were also inserted into the external jugular vein, the common carotid artery, and the left common hepatic vein. On the fourth day, measurements of cardiac output, arterial blood pressure, heart rate, inferior vena cava pressure, portal venous pressure, total hepatic blood flow, portal blood flow, and body weight were carried out in the fully conscious animals. The cecum was then ligated and after 3 more days, these measurements were repeated. In the septic high output state (in which the pig showed a higher cardiac output in the septic state than in the normal fasted state), the average cardiac output was 155% of that during normal fasting, the hepatic arterial flow increased 96%, the portal flow decreased 19%, the total hepatic flow increased 14%, the ratio of total hepatic blood flow to cardiac output decreased from 42% (normal fasted state) to 30%, and the total hepatic oxygen consumption increased 26%. In the low output state (in which the pig showed a lower cardiac output in the septic state than in the normal fasted state), the hepatic arterial flow decreased 74%, the portal flow decreased 23%, the total hepatic flow decreased 38%, the cardiac output decreased 38%, the ratio of total hepatic blood flow to cardiac output was 31%, total hepatic oxygen consumption decreased 29.1%, and splanchnic oxygen consumption decreased 22%. Only one of 11 pigs in the septic high output group died during the 3 days of observation, while 11 of 16 in the septic low output group died during this period. The remarkable difference in survival between the septic high and low output groups appeared to be largely due to the significant decrease in hepatic arterial flow and oxygen supply to the liver in the low output state.

5405 FORMATION OF COLONIES IN SOFT AGAR MEDIUM BY REGENERATING LIVER CELLS. *(E.)* Hays, D. M.; Sera, Y.; Koga, Y.; Hays, E. F. (Univ. South. California Sch. Med., Los Angeles). *Proc. Soc. Exp. Biol. Med.* 148(2):596-599, 1975.

The effect of serum factors, which have been shown to be stimulatory for marrow cells, on the colony-forming properties of cells from regenerating liver tissue was studied. Regenerating liver tissue was obtained from male Fischer rats and male C57BL/6 mice; this tissue was minced, trypsin-dispersed and cultured in soft agar medium. Designated cultures were supplemented with 40 µl of normal mouse serum (NMS), endotoxin mouse serum (EMS, obtained after the i.v. inoculation of endotoxin), or endotoxin rat serum (ERS). Normal liver cells, bone marrow cells from mature mice, and fetal rat and mouse liver cells were similarly treated. In the rat cell cultures, the greatest number of colonies was obtained from liver tissue obtained 48 hr after resection, the number of colonies being increased by a larger cell inoculum and being inversely related to the animal's weight. The effects of added EMS or ERS and the substitution of 15% human or rat serum for the normal 15% fetal calf serum additive were negligible. In the mouse cell cultures, colonies were obtained only from tissue obtained 72 to 168 hr postresection. These colonies were larger than those formed from regenerating rat liver. Only regeneration and fetal mouse liver formed a significant number of colonies in the absence of EMS or NMS; in the presence of these serum factors, the greatest number of colonies was formed by bone marrow, with fetal and regenerating liver yielding smaller and similar numbers of colonies. The cellular composition of these colonies was clearly distinguishable as granulocytic, granulocytic-macrophage, or macrophage. An additional colony type seen only in regenerating mouse liver cultures contained sheets of apparently epitheloid cells. The cell type of the rat liver colonies could not be clearly identified. The results indicate that regenerating mouse liver contains cells which can form granulocyte or macrophage colonies in soft agar in the presence of NMS or EMS. It appears that there is a nonhematopoietic cell population in regenerating liver that can also form colonies.

5406 ACTIVITIES OF HEPATIC MICROSOMAL ELECTRON TRANSPORT SYSTEM IN PROLONGED ETHANOL-TREATED RATS. *(E.)* Nakanishi, S.; Shiohara, E.; Tsukada, M.; Kinoshita, G. (Fac. Med., Shinshu Univ., Matsumoto, Japan). *Jpn. J. Pharmacol.* 25(1):71-73, 1975.

The activities of the main components of the hepatic microsomal electron transport system were studied in rats given prolonged ethanol treatment in the presence of aminopyrine or aniline. Wistar rats were given ethanol p.o. *ad libitum*, fasted 12 hr and sacrificed. Cytochrome P-450 and b_5 content, cytochrome P-450 reductase activity, cytochrome C reductase and NADPH oxidase activity, and microsomal protein content were determined. Cytochrome P-450 reductase, cytochrome C reductase, and NADPH oxidase activities increased significantly in the absence of drug substrate, compared to controls. Cytochrome P-450 reductase and cytochrome C reductase activities increased significantly in the presence of 3 mM of aminopyrine as compared to controls. With cytochrome P-450 reductase, there was no difference in the rate of reduction of the cytochrome in the presence of aminopyrine. The rate of cytochrome P-450 reduction was similar to that of controls in the presence of 2 mM of aniline. NADPH cytochrome C reductase activity was significantly higher than that of controls. The inhibitory effect of aniline was higher in the treated than in the control group. It is concluded that part of the cytochrome P-450 fraction increased by ethanol treatment is qualitatively different than that of controls.

5407 SYNCHRONIZED LIVER CELLS: A NEW TOOL FOR ANALYSIS OF CELL-CYCLE DEPENDENT CARCINOGEN-BINDING TO DNA IN VIVO. *(E.)* Rabes, H. M.; Iseler, G.; Tuczek, H. V.; Kerler, R. (Pathol. Inst., Univ. Munich, Germany). *Experientia* 31(6):686-687, 1975.

A qualitative comparison was made between the degree of DNA alkylation prior to, during and after DNA replication *in vivo*. Four groups of six Wistar rats each were 2/3 hepatectomized and given i.p. N,N-di(^{14}C)methyl-nitrosamine (0.18 Ci/mM), and ^{3}H-thymidine (0.8 Ci/g; 5 Ci/mM). The first group was partially hepatectomized prior to the injections. The second group was given the injections during continuous infusion of hydroxyurea, 28 hr after hepatectomy. The third group was given injections 43 hr after partial hepatectomy and four hr after stopping a hydroxyurea infusion (14 to 39 hr after hepatectomy); the fourth group was given the injections 47 hr after hepatectomy, and eight hr after stopping the infusion. Radioactivity from the two labels was measured separately by liquid scintillation spectrometry. The specific activity of DNA from the ^{3}H-thymidine was low in the G_1 phase, and also during hydroxyurea-induced DNA synthesis inhibition at 28 hr after hepatectomy. DNA synthesis showed a high rate 4 hr after removal of hydroxyurea blockage; it was reduced again by 8 hr after, when the cells were in G_2. The specific activity of ^{14}C in the G_1 phase and during hydroxyurea inhibition was low and without significant difference. DNA incorporation of ^{14}C increased significantly 4 hr after removal of hydroxyurea blockage. Specific activity remained at increased levels, even at 8 hr after, when the cells were in G_2. It is concluded that ^{14}C uptake varies with cell cycle phase, implying differing sensitivity to carcinogen-induced base alkylation.

5408 MICROFILAMENT DYSFUNCTION AS A POSSIBLE CAUSE OF INTRAHEPATIC CHOLESTASIS. *(E.)* Phillips, M. J.; Oda, M.; Mak, E.; Fisher, M. M.; Jeejeebhoy, K. N. (Toronto Gen. Hosp., Canada). *Gastroenterology* 69(1):48-58, 1975.

The possible function of pericanalicular microfilaments in the regulation of bile canalicular structure and function was investigated. Cultured rat hepatocytes, isolated perfused rat liver, and *in vivo* perfused rat liver were used as models. The cells were cultured with either cytochalasin B (20 µg/ml), 0.8% dimethylsulfoxide (DMSO) or phosphate buffer only (final concentration 0.0015 M). Samples were taken for electron microscopy at 1, 3, and 6 hr of incubation. Isolated liver was perfused for two hr, then 100 to 150 µg/ml of cytochalasin B and 0.2% DMSO were added to the perfusate. Bile was collected from the common duct and the volume was recorded hourly. Biopsies were taken 30 min, and 1, 2 and 3 hr after addition of the drug. In the *in vivo* experiment, bile flow was measured by common bile duct cannula for two hr. The liver was then perfused with 0.9% NaCl containing 100 µg/ml of cytochalasin B and 0.2% DMSO for one hr. Controls were given the NaCl with DMSO only. Biopsies were taken at 1, 2, and 3 hr after the start of perfusion. About 20-30% of the cultured hepatocytes were paired; they were joined by junctional complexes contiguous with the canaliculi. Light microscopy showed barely visible canicular luminal spaces in the controls, but widely dilated luminal spaces in the treated cells within 3-6 hr. Electron microscopy showed that the numbers of canalicular microvilli decreased steadily after 1 hr of treatment. In the isolated liver, light microscopy showed canalicular dilation, sinusoidal congestion, and fine hepatocytic vacuolarization. Electron microscopy 30 min after treatment showed dilated canaliculi, mostly in the periportal and midzonal regions, with microvillar loss. The pericanalicular ectoplasm was granular and amorphous. After 2 to 3 hr, the changes were panlobular. The cytoplasmic vacuoles contained mostly electron lucent material; the Golgi bodies were prominent. The overall cytoarchitecture was retained. Bile flow decreased over 75% after perfusion of a 100 µg/ml dose of the drug, and was virtually arrested after the 150 µg/ml dose. The *in vivo* liver did not show the cytoplasmic vacuoles and congestion of the isolated liver. Dilation of the canaliculi coincided with significant bile flow reduction. Microvilli were reduced, and autophagic vacuoles containing fibriliar and/or electron-dense material were seen in the panicanalicular area. One hr after treatment, bile flow was reduced 50% and nearly or completely arrested after 2 hr. It is concluded that the microfilaments maintain the canaliculi in a contracted or partially contracted state, and that their disruption is linked to bile flow reduction by an unknown mechanism.

5409 PLASMA DISAPPEARANCE OF RADIOACTIVITY AFTER INTRAVENOUS INJECTION OF LABELED BILE ACIDS IN MAN. *(E.)* Cowen, A. E.; Korman, M. G.; Hofmann, A. F.; Thomas, P. J. (Mayo Clin., Rochester, Minn.). *Gastroenterology* 68(6):1567-1573, 1975.

The kinetics of the disappearance of free and conjugated bile acids from plasma were studied. Each of six healthy volunteers received i.v. six bile acids (cholic and chenodeoxycholic acids and their glycine and taurine conjugates) in random order. The dose was 10 µCi for ^{14}C-labeled and 25 µCi for the ^{3}H-labeled bile acids. Blood samples were taken from the opposite antecubital vein to the point of injection at 2, 4, 6, 8, 10, 12, 15, 20, 30, 45, 60, 75, 90, 105, and 120 min after injection. The serum was separated and radioactivity was determined by liquid scintillation spectrometry. Subjects fasted throughout the experiment. Every primary bile acid, conjugated or unconjugated, had a biexponential plasma disappearance curve, indicating rapid disappearance. The half-life of the second component of the curve was about five times that of the first component, and they were highly correlated. The disappearance rates of cholylglycine and cholyltaurine were identical to each other, and were significantly faster than those of chenylglycine and chenyltaurine. For cholic and chenic acids, disappearance of the conjugated form was consistently faster than that of the unconjugated form. Dehydrocholyltaurine disappeared more slowly than any other acid; the half-life for the second exponential was 10 times that of the first. Cholic

acid, conjugated or not, showed considerably less protein binding in the serum than unconjugated chenodeoxycholic acid, deoxycholic acid or chenodeoxycholic conjugates. Differences in fractions bound to serum protein of cholic acid (91.0%), cholylglycine (85.6%) and cholyltaurine (82.8%) were statistically significant. It is concluded that the primary bile acids, conjugated or unconjugated, rapidly disappear from plasma in a biexponential fashion.

5410 REDUCED BILE FLOW IN RATS DURING SULFOBROMOPHTHALEIN INFUSION. *(E.)* Schulze, P. J.; Czok, G. (Pharmacol. Inst., Univ. Hamburg, Germany). *Toxicol. Appl. Pharmacol.* 32(2):213-224, 1975.

Male urethane-anesthetized Wistar rats with biliary fistulas were infused i.v. with sulfobromophthalein (BSP), or BSP-glutathione conjugate (BSP-GSH) (594 nM/100 g/min) for 100 min. With BSP infusion, the bile flow increased for 25 min and subsequently fell almost linearly. Biliary excretion of BSP-GSH correlated with the bile flow, whereas the hepatic concentration of this metabolite rose during infusion. The concentration of unconjugated BSP also increased in the liver. On the other hand, the infusion of BSP-GSH resulted in a long-lasting choleresis accompanied by high biliary concentrations of BSP-GSH without a concurrent increase in the concentration of BSP-GSH in the liver. The data indicate that during infusion of BSP, the biliary excretion of BSP-GSH is impaired after a peak at 30 min, although its hepatic content rises continuously to 10 μM/100 g body weight or 2.63 μM/g of liver, resp., after 100 min of infusion. The decrease in excretion of BSP-GSH was not due to the limited transport capacity for this metabolite, as a much higher rate of excretion was observed during BSP-GSH infusion. It is concluded that with the infusion of BSP the impaired biliary excretion of BSP-GSH and its increase in the liver are due to the presence of unconjugated BSP in the liver. It is suggested that the conjugation with glutathione may be the rate-limiting step in the biliary excretion of BSP.

5411 PHENOBARBITOL-ENHANCED BILIARY EXCRETION OF ADMINISTERED UNCONJUGATED AND CONJUGATED SULFOBROMOPHTHALEIN (BSP) IN THE RAT. *(E.)* Whelan, G.; Combes, B. (Univ. Texas Southwest. Med. Sch., Dallas). *Biochem. Pharmacol.* 24(13/14):1283-1286, 1975.

The mechanism by which phenobarbitol enhances biliary excretion of sulfobromophthalein (BSP) was studied in Sprague-Dawley rats given sodium phenobarbitol s.c. daily for seven days (7.5 mg/100 mg). Twenty-four hr after the last injection, the common bile duct was cannulized and either unconjugated BSP or synthetic BSP-glutathione was given i.v. (18 μM or 30 μM, resp./100g). Bile was collected 10 min prior to BSP injection and for four consecutive 10 min periods. A blood sample was taken from the aorta at the end of this time, and the liver was removed and weighed. BSP concentration in the bile was determined by spectrophotometry. Bile was also analyzed by descending chromatography on filter paper. Phenobarbitol-treated rats had a marked increase in liver size. Maximum dye excretion in controls after injection of unconjugated BSP was 1.34 μM/100 g/10 min. Phenobarbitol pretreatment significantly raised this to 2.06 μM/100 g/10 min. The rise was accompanied by a bile flow increase from 0.057 to 0.106 ml/100 g/10 min. Phenobarbitol did not affect maximal BSP bile concentration. Chromatography showed 56.8% of the excreted dye was conjugated in the controls and 77.7% in the treated group. Unconjugated dye excretion was not affected. Maximal excretion rates of the conjugated form were higher in the treated group (4.10 μM/100 g/10 min) than in controls (2.71 μM/100 g/10 min). The treated group also showed a rise in bile volume from 0.073 to 0.088 ml/100 g/10 min before conjugated BSP injection. Biliary BSP concentration was higher in rats given conjugated than unconjugated BSP, and was comparable in treated and control rats given conjugated BSP. It is concluded that phenobarbitol enhances BSP excretion by increasing transport of the conjugated form from hepatocytes to bile, and by increasing the rate of its intrahepatic metabolism.

5412 THE ACCUMULATION OF ASPARTATE IN THE PRESENCE OF ETHANOL IN RAT LIVER. *(E.)* Stubbs, M.; Krebs, H. A. (Radcliffe Infirm., Oxford, England). *Biochem. J.* 150(1):41-45, 1975.

The ability of ethanol to cause accumulation of aspartate in rat liver in the presence of excess nitrogen was investigated. Hepatocytes from Wistar rats were isolated and put into suspension; in some cases the cytosolic and mitochondrial components were separated and labeled with markers. The cell suspension was analyzed for glucose, lactate, pyruvate, aspartate, glutamate, alanine, NH_4^+, urea, glutamine, malate, and 2-oxoglutarate content. Ethanol had no effect on aspartate yield in the suspension; it caused an increase in perfused liver. Of the total nitrogen, 17.5% appeared as NH_4^+ in suspension, as compared to 64% in perfused liver. Addition of 1.5 U/flask of urease to the hepatocyte suspension caused a 7-fold increase in aspartate formation on addition of ethanol, indicating that aspartate formation was dependent on NH_4^+ concentration. In the presence of 10 mM of lactate and 10 mM of NH_4Cl, ethanol caused a 22-fold increase in aspartate concentration, and inhibited glucose synthesis by 73%. Ethanol did not affect the amount of NH_4^+ removal, but increased the amount of NH_4^+ converted to aspartate and glutamate, and decreased the amount converted to urea. There was no increase in yields of aspartate and glutamate with or without ethanol, lactate or NH_4Cl. Addition of ornithine increased urea synthesis with or without the presence of ethanol, and abolished aspartate accumulation. It also depressed glucose synthesis from lactate and NH_4Cl, but ethanol partially overcame this depression. When the cells were fractionated, ethanol increased the aspartate yield 10-fold in the cytosol fraction, but did not affect the mitochondrial fraction. It also increased the glutamate content by 1.9 times and 3.7 times in these fractions, resp. The 2-oxoglutarate content was halved by ethanol in the cytosol, but was unaffected in the mitochondria. It is concluded that accumulation of aspartate in the presence of ethanol depends on high concentrations of NH_4^+.

5413 BIOCHEMICAL STUDIES ON THE EFFECT OF A DEPROTEINIZED BLOOD DERIVATIVE ON THE RECOVERY OF RAT LIVER AFTER HEMORRHAGIC SHOCK LASTING 60 MINUTES. *(Ger.)* Wolff, R. (Steglitz Clin., Free Univ., Berlin, Germany). *Arzneim. Forsch.* 25(3):388-392, 1975.

The effect of Actihaemyl, a deproteinized extract of calf blood, was tested for its effect on hepatic anoxia caused by hemorrhagic shock. Ether-anesthetized male Wistar rats were bled until their average blood pressure had decreased to 40 mm Hg. After 60 min, their blood was retransfused along with Actihaemyl (5 ml/kg) or placebo (3.51% NaCl solution). Rats were sacrificed 30-120 min after retransfusion, and their livers were removed and homogenized. During shock significant increases occurred in lactate β-hydroxybutyrate contents with decreases in pyruvate values. Anoxia was characterized by an increase in the lactate/pyruvate ratio and a decrease in the ATP/ADP ratio. The glycogen content of the liver decreased to extremely low values. The concentrations of all metabolites studied, with the exception of ADP and acetoacetate, depended on the recovery time after hemorrhagic shock in both rats treated with Actihaemyl and in the controls. In treated rats lactate and β-hydroxybutyrate contents decreased more rapidly than in controls so that the lactate/pyruvate and β-hydroxybutyrate/acetoacetate ratios were decreased. These findings suggest that Actihaemyl increases O_2 uptake in the liver following hemorrhagic shock.

5414 THE EFFECT OF ALCOHOL ON LIPID METABOLISM IN THE ISOLATED PERFUSED RAT LIVER. *(Ger.)* Ast, E.; Durr, C.; Bauer, E.; Papenberg, J. (Med. Clin., Univ. Heidelberg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:479-481, 1974.

The synthesis and secretion of lipoproteins were studied in the presence and absence of ethanol in isolated perfused livers from nonfasted rats. Livers were perfused for 4 hr with a medium containing 127 mM of oleic acid bound to albumin and 30 μCi of ^{14}C-leucine; 4 mM of ethanol were added at the beginning and an additional 3 mM were added 120 min later to experimental, but not to control, perfusates. In the presence of ethanol, secretion of total very low density lipoproteins (VLDL) into the medium increased significantly from 1099 to 1686 μg/g of liver, primarily due to increased triglyceride secretion. The specific activity of apoprotein in the VLDL fraction increased significantly from 122 to 247 cpm/μg in the presence of ethanol. Ethanol had no effect on low- or high-density lipoprotein fractions. Triglyceride concentrations in perfusion medium increased significantly from 2498 to 4203 μg/g of liver, and fatty acid concentrations increased from 2.43 to 3.52 mM/ml. The total lipid content of the liver decreased by 8.55 mg/g of liver in controls but increased significantly by 6.16 mg/g of liver in the presence of ethanol. Differences between the triglyceride and cholesterol contents of the liver were also significant, but no appreciable difference was observed for phospholipids. After 4 hr of perfusion, the fatty acid uptake of the liver was significantly lower in the presence of ethanol (34 mM/g) than in its absence (44 mM/g). Thus, these ethanol-induced changes in lipid metabolism are similar to ethanol-induced type IV hyperlipoproteinemia in man, which results mostly from increases in free fatty acids.

5415 SIMILARITY OF ARTERIAL AND INTRAVENOUS VASOPRESSIN ON PORTAL AND SYSTEMIC HEMODYNAMICS. *(E.)* Barr, J. W.; Lakin, R. C.; Roesch, J. (Univ. Oregon Health Sci. Cent., Portland, Ore.). *Gastroenterology* 69(1):13-19, 1975.

The effects of superior mesenteric arterial and i.v. infusions of vasopressin and low- and high-dose i.v. infusions of vasopressin on splanchnic and systemic hemodynamics were compared in 20 anesthetized dogs. Injections of 5-10 g of microspheres into the portal circulation in five dogs of the first group (10 dogs) resulted in an immediate increase of portal pressure from a mean of 7.1 to 14.8 mm Hg. This level of increase lasted for 5-10 min after which portal pressure gradually fell and stabilized at a mean of 10.7 mm Hg. Comparison of selective arterial and i.v. infusions showed no statistically significant difference between the degree of changes in portal flow, portal and systemic blood pressure, and cardiac output. Only the superior mesenteric artery flow showed a greater decrease with the selective arterial injection. In the second group of 10 dogs, infusion of 14.0 mU/kg/min caused sharp falls in the superior mesenteric artery flow, portal vein flow and portal pressures, which reached their lowest levels in about 3-5 min. After the infusion, all parameters returned to normal levels, reaching the base line at the mean period of 55 min. Infusion of 2.75 mU/kg/min caused gradual decreases (about 10 min) and resulted in a maximal fall in superior mesenteric artery flow to 51% base line, in portal flow to 56% base line, and in portal pressures to 73% base line. The mean period of return to normal was 35 min. The applicability of these findings in dogs with normal or only slightly increased portal pressure to patients with liver disease and portal hypertension can only be established by clinical trials. The findings indicate that an easily applied i.v. infusion of vasopressin could again become the primary method for vasoconstrictive therapy of massive bleeding from varices.

5416 ACUTE LIVER INJURY BY VINYL CHLORIDE: INVOLVEMENT OF ENDOPLASMIC RETICULUM IN PHENOBARBITAL-PRETREATED RATS. *(E.)* Reynolds, E. D.; Jaeger, R. J.; Murphy, S. D. (Peter Bent Brigham Hosp., Boston, Mass.). *Environ. Health Perspect.* 11:227-233, 1975.

An ultrastructural description of acute liver injury by vinyl chloride in male Holtzman rats pretreated with phenobarbital is presented. Daily consumption of phenobarbital was approximately 10 mg/100 g rat. Treated and nontreated rats were exposed to 5% vinyl chloride for 6 hr once or to 5% vinyl chloride for 6 hr each on five consecutive days. Twenty-four hr after the onset of a single exposure of 5% vinyl chloride, there was diffuse vacuolization of the

cytoplasm of centrilobular liver parenchyma and focal areas of necrosis of midzonal parenchyma in phenobarbital-pretreated rats. Vacuolization involved approximately two-thirds of the hepatic parenchyma, and midzonal necrosis was focal throughout most of the liver and became extensive and confluent toward the dorsal aspect. In contrast, livers of animals exposed to vinyl chloride without phenobarbital pretreatment appeared normal. Livers of phenobarbital-pretreated animals exposed to 5% vinyl chloride on five consecutive days contained broad tracts of stroma depleted of parenchymal cells which correspond in distribution and extent to the areas of necrosis seen at 24 hr. Vacuolated liver parenchymal cells in phenobarbital-pretreated rats killed 24 hr after the onset of exposure to 5% vinyl chloride revealed extensive dilation of the cysternae of the rough endoplasmic reticulum and coalescence of the smooth into discrete aggregates of smooth surfaced tubules conspicuously flecked with areas of increased electron opacity. The ultrastructural observations that the acute liver lesion produced by vinyl chloride primarily involves components of the endoplasmic reticulum, coupled with the fact that phenobarbital enhances injury, suggest that this organelle is the primary site of generation of toxic metabolites from vinyl chloride.

5417 ALPHA FETOPROTEIN: EFFECT OF HETEROLOGOUS ANTISERUM ON HEPATOMA CELLS IN VITRO. *(E.)* Mizejewski, G. J.; Young, S. R.; Allen, R. P. (Div. Lab. Res., New York State Dep. Health, Albany). *J. Natl. Cancer Inst.* 54(6):1361-1367, 1975.

The cytotoxic effect of rabbit antisera against mouse alpha-fetoprotein (AFP) on mouse hepatoma cells was studied. Mouse BW7756 hepatoma cells and C57L/J muscle fibroblasts were grown in culture to near confluency, washed, and grown in Eagle's minimum essential medium-Hanks' balanced salt solution containing either 4% immunized (anti-AFP) or 4% non-immunized rabbit serum. Cytotoxic antibody assays were also carried out on fresh tumor cell suspensions. The BW7756 cells synthesized AFP *in vitro*, the AFP being immunologically identical to that circulating in the sera of hepatoma-bearing mice. In the cytotoxicity assays, the presence of intact and/or inactivated anti-AFP serum inhibited the growth of the AFP-producing cells. The cytotoxic effects of the antiserum depended on both exposure time and serum concentration. The cytotoxicity was complement independent, as demonstrated by studies with heat-deactivated serum devoid of extrinsic complement. Specificity of the antisera for the target cells was demonstrated by absorption with purified mouse AFP. The results could be explained by the postulated presence of AFP on the hepatoma cell surface.

5418 INFLUENCE OF SUBTOTAL HEPATECTOMY ON PEROXISOMES AND PEROXISOMAL ENZYMES OF RAT LIVER AND ISOLATED LIVER CELL FRACTIONS. *(E.)* Goldenberg, H.; Heuttinger, M.; Boeck, P.; Kramar, R. (Dep. Med. Chem., Univ. Vienna, Austria). *Histochemistry* 44(1):44-47, 1975.

The influence of subtotal hepatectomy on peroxisomes and peroxisomal enzymes of rat liver and isolated liver cell fractions was investigated in Sprague-Dawley rats. The activities of peroxisomal enzymes were followed one to ten days after subtotal (60-70%) hepatectomy in homogenates prepared from regenerating livers and in cell fractions isolated from them. Catalase activity was depressed in the total liver homogenate as well as in the mitrochondrial and soluble fractions; it did not change appreciably in the microsomal and lysomal fractions. α-Hydroxyacid oxidase behaved in a similar fashion. In contrast to these enzymes, urate oxidase activity remained unchanged in total liver homogenate, whereas it decreased in mitrochondrial and increased in lysosomal fractions and microsomal fractions during the first five days after operation. These results agree well with the assumption that microbody proliferation is initiated by the fragmentation of large peroxisomes. Biochemical examinations were paralleled by morphological and histochemical studies. The number of peroxisomes increased during the first day after partial hepatectomy, and this was accompanied by a decrease in particle size. During the first mitotic wave (24-36 hr postoperative), the number of peroxisomes/cell was reduced by about half. After this time, the number and size of the particles began to increase. Positive staining of ribosomes was frequently observed in the vicinity of peroxisomes after the application of the cytochemical catalase reaction (alkaline diaminobenzidine medium). This phenomenon is interpreted to represent a diffusion artifact rather than the cytochemical identification of newly synthesized catalase.

5419 INFLUENCE OF PHOSPHOLIPIDS ON LIVER DAMAGE. I. CARBON TETRACHLORIDE POISONING AND ALTERATIONS IN AMINO ACID UPTAKE, PEROXIDATION, SIALIC ACID CONTENT, AND LYSOSOMAL ENZYMES. *(E.)* Gerber, G. B.; Bartsch, G. G.; Deroo, J. (Dep. Radiat. Biol., d'Centre Etude Nucleaire, Mol, Belgium). *Acta Hepatogastroenterol. (Stuttg.)* 22(3):175-180, 1975.

Changes in peroxidation, amino acid uptake [α-amino isobutyrate], sialic acid content and lysosomal enzymes were investigated in male Wistar rats and male BALB/c^+ mice after carbon tetrachloride poisoning and treatment with phospholipids. The animals received 1 ml of a 10% phospholipid suspension for 7 days prior to administration of CCl_4 by stomach tube. The labeled α-amino isobutyrate (1 μCi) was injected i.p. 1 hr or 18 hr before sacrifice. CCl_4 was given 6 or 24 hr before sacrifice. Homogenates were made of liver, muscle, heart and brain tissue. The uptake and retention of α-amino isobutyrate in liver increased markedly during CCl_4 poisoning. Phospholipids did not alleviate this effect and seemed to delay release of α-amino butyrate by liver. In CCl_4 poisoned rats, the uptake of α-amino butyrate was diminished in the heart but enhanced in the brain. No changes were seen in muscle. Total sialic acid in liver increased 24 hr after administration of CCl_4. Free sialic acid increased slightly in liver of CCl_4 treated rats and decreased in the brain of CCl_4 and phospholipid treated animals. Peroxides and peroxide formation were determined by the thiobarbituric acid

reaction. Preformed peroxides were not affected by CCl_4 poisoning but they were diminished by phospholipid treatment. Peroxide formation was significantly greater in rats not receiving phospholipids and was markedly increased 24 hr after CCl_4 poisoning. More preformed peroxides were present in mouse than in rat liver. Peroxide formation was enhanced in mouse liver poisoned with CCl_4, but protection with phospholipids had little effect. Lysosomal enzymes were slightly influenced by phospholipid treatment and/or CCl_4 poisoning, except for a slight increase in free catheptic activity in CCl_4 poisoned mouse liver. The observed increase in catalytic peroxide formation, as well as the higher levels of peroxides in poisoned mouse liver early after intoxication, suggests that peroxides participate in the hepatotoxicity of CCl_4. Peroxidation appears to be the primary symptom of CCl_4 intoxication, with change in protein synthesis only a secondary effect.

5420 THE RELATION BETWEEN LIVER CIRCULATION AND LIVER FUNCTION: ITS CLINICAL RELEVANCE. *(Ger.)* Preisig, R. (Inst. Clin. Pharmacol., Univ. Bern. Switzerland). *Z. Gastroenterol.* 13(suppl.):104-112, 1975.

5421 BILIRUBIN METABOLISM. *(Ger.)* Teichmann, W. (Med. Clin., Univ. Rostock, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):115-118, 1974.

5422 SHOULD A FATTY LIVER BE REGARDED AS A PREVIOUSLY DAMAGED ORGAN? LIVER REGENERATION FOLLOWING PARTIAL HEPATECTOMY IN NORMAL AND OBESE MICE. *(Ger.)* Grimmel, K.; Paulini, K.; Rakow, L.; Beneke, G. (Ctr. Biol. Theoretical Med., Univ. Ulm, Germany). *Med. Welt* 25(46):1930-1934, 1974.

5423 ETHANOL METABOLISM AND LIVER DAMAGE IT PRODUCES. *(Ger.)* Schmidt, L. (Jewish Hosp., Berlin, Germany). *Ther. Ggw.* 113(11): 1911-1920, 1974.

5424 SODIUM GLYCO-24,25-DIHYDROFUSIDATE. BILIARY EXCRETION AND EFFECT ON BILE LIPID SECRETION IN THE RAT. *(Fr.)* Montet, J. C.; Montet, A. M.; Amic, J.; Hauton, J. C. (Res. Group Lipid Transport, Marseille, France). *Biol. Gastroenterol. (Paris)* 7(4):326, 1974.

5425 ROLE OF BILE AND PANCREATIC SECRETIONS IN MUCOSAL GROWTH AFTER JEJUNECTOMY IN RATS FED CHOW OR PREDIGESTED DIET. *(E.)* Weser, E.; Tawil, T. (Univ. Texas Health Sci. Cent., San Antonio, Tex.). *Gastroenterology* 68(4):1012, 1975.

5426 EFFECT OF THYROID FUNCTION ON THE ELECTROPHORETIC PATTERN OF MITOCHONDRIAL PROTEINS IN RAT LIVER. *(Fr.)* Baudry, M.; Clot, J. P.; Michel, R. (Teach. Res. Unit Pharm. Sci. Biol., Paris, France). *Biochimie* 57(1):77-83, 1975.

5427 NUCLEIC ACIDS IN THE CEREBRAL CORTEX AND LIVER FOLLOWING ISOLATED COMPLETE ANOXIA OF THE CENTRAL NERVOUS SYSTEM. *(Rus.)* Kozhura, V. L. (Lab. Exp. Physiol. Resuscitation, Moscow, USSR). *Biull. Eksp. Biol. Med.* 79(2):34-35, 1975.

5428 CIRCADIAN RHYTHM IN HEPATIC AND INTESTINAL BILE SALT SYNTHESIS IN THE RAT. EFFECT OF BILE ACIDS. *(Fr.)* Reynier, M. O.; Mule, A.; Crotte, C.; Gerolami, A. (Unit Res. Gastrointestinal Dis., INSERM, Marseille, France). *Biol. Gastroenterol. (Paris)* 7(4):325, 1974.

5429 SOME FINDINGS ON THE ASSOCIATION OF LECITHIN AND POLYPEPTIDES IN THE BILE. *(Fr.)* Lafont, H.; Domingo, N.; Lairon, D.; Hauton, J. C. (Res. Group Lipid Transport, Marseille, France). *Biol. Gastroenterol. (Paris)* 7(4):328, 1974.

5430 EFFECT OF GLYCEROL ON THE SYNTHESIS OF VERY LOW DENSITY LIPOPROTEINS BY THE LIVER. *(Fr.)* Raisonnier, A.; Petit, D.; Infante, R. (St. Antoine Hosp., Paris, France). *Biol. Gastroenterol. (Paris)* 7(4):738, 1974.

5431 ASPIRIN-INDUCED CHOLERESIS IN THE DOG. *(Fr.)* Bienfait, D.; Dumont, M.; Duval, M.; Poupon, R.; Erlinger, S. (Beaujon Hosp., Clichy, France). *Biol. Gastroenterol. (Paris)* 7(4):323-324, 1974.

5432 EFFECT OF INGESTING HEATED FLAXSEED OIL ON SERUM ALKALINE PHOSPHATASE AND TRANSAMINASE ACTIVITIES IN THE RAT. *(Fr.)* Grandgirard, A.; Loisel, W. (Res. Stn. Quality Hum. Foods, Dijon, France). *Ann. Nutr. Aliment* 28(3):121-133, 1974.

5433 EFFECT OF INGESTING HEATED FLAXSEED OIL ON THE DIGESTION AND COMPOSITION OF FATTY ACIDS IN HEPATIC AND ADIPOSE TISSUE LIPIDS IN THE GROWING MALE RAT. *(Fr.)* Potteau, B. (Res. Stn. Quality Hum. Foods, Dijon, France). *Ann. Nutr. Aliment* 28(3):135-158, 1974.

5434 MECHANISMS INVOLVED IN BILE SECRETION IN MAN. *(Ger.)* Kuska, J. (Silesian Acad. Med., Katowice, Poland). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):145-150, 1974.

5435 STUDIES IN THE COMPOSITION OF LIVER TRIGLYCERIDES AND TRIGLYCERIDES RECOVERED FROM THE CIRCULATION AFTER LAURIC ACID PERFUSION OF ISOLATED RAL LIVER. *(Fr.)* Chanussot, F.; Bezard, J.; Debry, G. (Dep. Nutr. Metab. Dis., Univ. Nancy, France). *Ann. Nutr. Aliment.* 28(5):407-435, 1974.

5436 THE EFFECT OF LOADING HEPATIC LYSOSOMES WITH TRITON WR 1339 ON THE DEVELOPMENT OF CHRONIC TOXIC HEPATITIS. *(Rus.)* Korolenko, I. A.; Titova, V. G.; Drobrovolskaia, S. G.; Iakobson, G. S. (Novosibirsk Med. Inst., USSR). *Biull. Eksp. Biol. Med.* 79(1):21-23, 1975.

5437 THE EFFECT OF OXYGEN DEFICIENCY ON THE CONTENT OF SOLUBLE PROTEINS IN THE BRAIN AND LIVER OF RATS AT VARIOUS TEMPERATURES. *(Rus.)* Gasteva, S. V.; Chetverikov, D. A.; Shmelev, A. A. (I. P. Pavlov Inst. Physiol., Leningrad, USSR). *Biull. Eksp. Biol. Med.* 79(2):36-37, 1975.

5438 CHANGES IN THE GENERAL HEPATIC CIRCULATION AT THE EARLY POSTRESUSCITATION PERIOD. *(Rus.)* Evtushenko, A. Ya. (Kemercvo Med. Inst., USSR). *Biull. Eksp. Biol. Med.* 79(2):14-17, 1975.

5439 BILIRUBIN METABOLISM. I: ORIGIN OF BILIRUBIN. CIRCULATION OF BILIRUBIN IN THE PLASMA. *(Sp.)* Candel Monserrate, I. (Complutense Univ., Madrid, Spain). *Rev. Esp. Enferm. Apat. Dig.* 45(5):329-344, 1975.

5440 THE LIVER AND DRUG METABOLISM. *(Fr.)* Darnis, F. (Beaujon Hosp., Clichy, France). *Cah. Med.* 15(10):637-643, 1974.

5441 THE DENATURATION OF SERUM GLUTAMATE-OXALOACETATE TRANSAMINASE (SGOT) AND ITS RELATION TO THE GENOTYPE AND PERFORMANCE OF SWINE. *(Ger.)* Kintscher, M.; Hartleb, K. (no affil.). *Wissenchaftl. Z. Humboldt Univ.* [*Math.-Nat. R*] 23(4):437-446, 1974.

5442 EFFECT OF PHENOBARBITAL ON THE COMPOSITION OF BILE IN MAN. *(Fr.)* Capron, J. P.; Dumont, M.; Dairou, R.; Erlinger, S. (Beaujon Hosp., Clichy, France). *Biol. Gastroenterol. (Paris)* 7(4):324, 1974.

5443 EFFECT OF GENTAMYCIN ON THE ENERGY METABOLISM OF RAT MITOCHONDRIA IN THE LIVER AND KIDNEYS. *(Fr.)* Bendirdjian, J. P.; Foucher, B.; Rollin, P.; Fillastre, J. P. (Res. Group Cell Physiol. Pathol., Univ. Rouen, France). *C. R. Acad. Sci.* [*D*] *(Paris)* 280(12):1489-1492, 1975.

5444 ACUTE THIOACETAMIDE-INDUCED LIVER INJURY: NEUROPHYSIOLOGICAL CHANGES IN CATS. *(Ger.)* Fischer, B.; Holm, E.; Reith, H. I.; Sommer, U.; Urbanek, U. (Fac. Clin. Med., Univ Heidelberg, Mannheim, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80: 439-443, 1974.

5445 THE EFFECT OF SECRETIN ON BILE FLOW AND HEPATIC BLOOD FLOW. *(Ger.)* Brunner, H.; Slat, B.; Kretschmer, G.; Funovics, J. (Dep. Gastroenterol., Univ. Vienna, Austria). *Verh. Dtsch. Ges. Inn. Med.* 80:435-436, 1974.

5446 CHANGES IN ^{125}I-FIBRINOGEN AND ^{131}I-ALBUMIN IN EXPERIMENTAL GALACTOSAMINE HEPATITIS. *(Ger.)* Von Mahn, I.; Merkel, H.; Sattler, E. L.; Muller-Berghaus, G. (Cent. Intern. Med., Univ Giessen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:437-439, 1974.

5447 CHANGES IN THE ACTIVITIES OF ENZYMES LOCATED AT DIFFERENT SITES IN THE CELL FOLLOWING HETEROTOPIC AUXILIARY LIVER TRANSPLANTATION IN RATS. *(Ger.)* Bode, C.; Paidlick, A.; Zelder, O.; Jerusalem, C. R.; Bode, J. C. (Med. Clin., Univ. Marburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:427-429, 1974.

5448 α-NAPHTHYLISOTHIOCYANTE-INDUCED CHOLESTASIS IN THE RAT LIVER: EFFECT ON GLUCOCORTICOID ADMINISTRATION ON THE METABOLITES OF CARBOHYDRATE METABOLISM. *(Ger.)* Wolfert, W.; Ohnemus, H.; Goebell, H. (Cent. Intern. Med. Pediatr., Univ. Ulm, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:432-435, 1974.

5449 DETECTION OF A CELL COAT IN HEPATOCYTES IN MONOLAYER CULTURE WITH THE AID OF THE PHYTOHEMAGGLUTININ CONCANAVALLIN. *(Fr.)* Charbonne, F.; Perissel, B.; Padieu, P.; Turchini, J. P. (Fac. Med., Clermont-Ferrand, France). *C. R. Acad. Sci.* [*D*] *(Paris)* 280(13):1583-1585, 1975.

5450 THE EFFECT OF A FOOD ADDITIVE, THE ANTIOXIDANT DI-tert.-BUTYLHYDROXY-TOLUENE (BHT), ON ELECTRON TRANSPORT AND THE COUPLING OF OXIDATION AND PHOSPHORYLATION IN RAT LIVER MITOCHONDRIA. *(Fr.)* Pascal, G.; Terroine, T. (Nat. Inst. Agron. Res., Jouy-en-Josas, France). *C. R. Acad. Sci.* [*D*] *(Paris)* 280(15):1833-1836, 1975.

5451 HYDROXYLATION OF DERIVATIVES OF ANILINE AND AMINOANTIPYRINE (1-PHENYL-2,3-DIMETHYL-5-AMINOPYRAZOLONE) IN LIVER ENDOPLASMATIC RETICULUM. *(Rus.)* Archakov, I. I.; Karuzina, I. I.; Tveritinov, V. N.; Kokareva, N. S. (N. I. Pirogov 2nd State Med. Inst. Moscow, USSR). *Biokhimiia* 40(1):32-39, 1975.

5452 PROSTAGLANDIN E_1: ANOMALOUS EFFECTS ON GLUCOSE PRODUCTION IN RAT LIVER. *(E.)* Wheeler, G. E.; Epand, R. M. (Health Sci. Cent., Hamilton, Canada). *Mol. Pharmacol.* 11(3):335-339, 1975.

5453 DETERMINATION OF DICUMAROL METABOLITES IN BILE OF RATS. *(E.)* Conway, W. D.; Lee, F. H.; Neufeld, L. (Sch. Pharm., State Univ. New York, Buffalo). *J. Pharm. Sci.* 64(7):1158-1162, 1975.

5454 THE METABOLISM OF SOME FOLATES IN THE RAT. *(E.)* Beavon, J. R. G.; Blair, J. A. (Dep. Chem., Univ. Aston, Birmingham, England). *Br. J. Nutr.* 33(3):299-308, 1975.

5455 VITAMIN B_{12} NUTRITION AND METABOLISM IN THE BABOON (Papio cynocephalus). *(E.)* Siddons, R. C.; Jacob, F. (Wellcome Trust Res. Lab., Nairobi, Kenya). *Br. J. Nutr.* 33(3):415-424, 1975.

5456 THE INFLUENCE OF ISO-ENERGETIC AMOUNTS OF SUCROSE AND STARCH ON SERUM ALBUMIN CONCENTRATIONS AND LIVER COMPOSITION OF WEANLING RATS GIVEN A LOW-PROTEIN DIET. *(E.)* Grimble, R. F.; Dickerson, J. W. T. (Dep. Biochem., Univ. Surrey, England). *Nutr. Metabol.* 17(5):272-281, 1974.

5457 IDENTIFICATION OF ORNITHINE AND ARGININE CONJUGATES OF CHOLIC ACID BY MASS SPECTROMETRY. *(E.)* Myher, J. J.; Marai, L.; Kuksis, A.; Yousef, I. M.; Fisher, M. M. (Banting Best Dep. Med. Res., Toronto, Canada). *Can. J. Biochem.* 53(5):583-590, 1975.

5458 RIBOSOME ACCUMULATION IN 3-METHYLCHOLANTHRENE-INDUCED LIVER GROWTH IN ADULT MALE RATS. *(E.)* Argyris, T. S.; Heinemann, R. (Upstate Med. Cent., Syracuse, N.Y.). *Exp. Mol. Pathol.* 22(3):335-341, 1975.

5459 CONJUGATED METABOLITES OF ESTRIOL IN RAT BILE. *(E.)* Nambara, T.; Kawarada, Y. (Pharm. Inst., Tohoku Univ., Sendai, Japan). *Chem. Pharm. Bull.* 23(3):698-700, 1975.

5460 NUCLEAR PROTEIN CHANGES IN RAT HEPATOMAS CORRELATING WITH GROWTH RATE. *(E.)* Lea, M. A.; Koch, M. R.; Morris, H. P. (New Jersey Med. Sch., Newark). *Cancer Res.* 35(7):1693-1697, 1975.

5461 THERMAL AND PHOTOCHEMICAL REACTIONS OF BILIRUBIN IX-α. *(E.)* McDonagh, A. F. (Dep. Med. Pharm. Chem., Univ. Calif., San Francisco). *Ann. N.Y. Acad. Sci.* 244:553-569, 1975.

5462 ON MECHANISMS IN HYPERAMMONEMIC COMA--WITH PARTICULAR REFERENCE TO HEPATIC ENCEPHALOPATHY. *(E.)* Hindfelt, B. (Dep. Neurol., Univ. Hosp., Lund, Sweden). *Ann. N.Y. Acad. Sci.* 252:116-123, 1975.

5463 THE ENVIRONMENT OF FUNCTION OF LIVER AND RED BLOOD CELLS. *(E.)* Drabkin, D. L. (Dep. Biochem., Univ. Pa., Philadelphia). *Ann. N.Y. Acad. Sci.* 244:603-623, 1975.

5464 STUDIES ON THE SH⇌SS EQUILIBRIUM IN BIOLOGICAL MEDIA. V. ON THE DETERMINATION OF SULFHYDRYL GROUPS IN TISSUES. *(E.)* Olinescu, R.; Uleu, C. (Clin. Hosp. Fundoni, Bucharest, Romania). *Rev. Roum. Biochim.* 11(4):283-289, 1974.

5465 IN VIVO ASSESSMENT OF LIVER SIZE IN THE RAT. *(E.)* Leach, K. G.; Karran, S. J.; Wisbey, M. L.; Blumgart, L. H. (Welsh Nat. Sch. Med., Cardiff, Wales, U.K.). *J. Nucl. Med.* 16(5):380-385, 1975.

5466 T-CELL DEPENDENCE OF IMMUNE RESPONSE TO HEPATITIS B ANTIGEN IN MICE. *(E.)* Roberts, I. M.; Bernard, C. C.; Vyas, G. N.; Mackay, I. R. (Walter Eliza Hall Inst. Med. Res., Victoria, Australia). *Nature* 254(5501):606-607, 1975.

5467 AN IMPROVED NONSUTURE METHOD FOR PORTACAVAL ANASTOMOSIS IN THE RAT. *(E.)* Funovics, J. M.; Cummings, M. G.; Shuman, B. A. L.; James, J. H.; Fischer, J. E. (Mass. Gen. Hosp., Boston). *Surgery* 77(5):661-664, 1975.

5468 METABOLISM OF LEUCINE AND ALANINE IN GROWING RATS FED THE DIETS WITH VARIOUS PROTEIN TO ENERGY RATIOS. *(E.)* Tanaka, H.; Yamaguchi, M.; Kametaka, M. (Dep. Anim. Sci. Agric. Chem., Univ. Tokyo, Japan). *Agric. Biol. Chem.* 39(2):507-514, 1975.

5469 SOME ASPECTS OF THE BIOSYNTHESIS OF RIBOSOMAL STRUCTURAL PROTEINS IN REGENERATING RAT LIVER. *(E.)* Ogata, K.; Tsurugi, K.; Nabeshima, Y. (Niigata Univ. Sch. Med., Japan). *Acta Biol. Med. Ger.* 33(5-6):963-969, 1974.

5470 THE HORMONAL CONTROL OF GLUCONEOGENESIS BY REGULATION OF MITOCHONDRIAL PYRUVATE CARBOXYLATION IN ISOLATED RAT LIVER CELLS. *(E.)* Garrison, J. C.; Haynes, Jr., R. C. (Univ. Va. Sch. Med., Charlottesville). *J. Biol. Chem.* 250(8):2769-2777, 1975.

5471 INTRACELLULAR LOCALIZATION OF THE 3-HYDROXY-3-METHYLGLUTARYL COENZYME A CYCLE ENZYMES IN LIVER. SEPARATE CYTOPLASMIC AND MITOCHONDRIAL 3-HYDROXY-3-METHYLGLUTARYL COENZYME A GENERATING SYSTEMS FOR CHOLESTEROGENESIS AND KETOGENESIS. *(E.)* Clinkenbeard, K. D.; Reed, W. D.; Mooney, R. A.; Lane, M. D. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *J. Biol. Chem.* 250(8):3108-3116, 1975.

5472 IN VIVO INCORPORATION OF CYTIDINE-MONOPHOSPHATE-CILIATINE INTO RAT LIVER LIPIDS. *(E.)* Tamari, M.; Cassaigne, A.; Lacoste, A. M.; Nevzil, E. (Biochem. Lab., Univ. Bordeaux II., France). *Biochimie* 57(1):97-103, 1975.

5473 LIVER CHANGES IN PIGS FED A DIET LOW IN PROTEIN AND HIGH IN FAT AND CHOLESTEROL. *(E.)* Gupta, P. P.; Tandon, H. D.; Ramalingaswami, V. (All-India Inst. Med. Sci., New Delhi, India). *Indian J. Med. Res.* 62(11):1659-1672, 1974.

5474 STUDIES ON METABOLISM IN RATS WITH ETHIONINE-INDUCED FATTY LIVER. *(E.)* Ramakrishna Rao, P.; Ramakrishnan, S. (Jawaharal Inst. Post-Grad. Med. Educ., Pondicherry, India). *Indian J. Biochem. Biophys.* 11(3):252-255, 1974.

5475 SUBCELLULAR DISTRIBUTION OF PYROPHOSPHATE PHOSPHOHYDROLASE IN THE RAT LIVER. *(E.)* Dumitru, I. F.; Filipescu, G.; Niculescu, S.; Iordachescu, D.; Balteanu, E. (Dep. Biochem., Inst. Biol. Sci., Bucharest, Romania). *Rev. Roum. Biochim.* 11(3):167-192, 1974.

5476 THE ACTION OF REDUCED GLUTATHIONE ON TRYPTOPHAN OXYGENASE. *(E.)* Olinescu, R.; Pascu, E. (Clin. Hosp. Fundeni, Bucharest, Romania). *Rev. Roum Biochim.* 11(3):185-192, 1974.

5477 IN VITRO METABOLISM OF POLYCHLORINATED BIPHENYLS-^{14}C. *(E.)* Greb, W.; Klein, W.; Coulston, F.; Golberg, L.; Korte, F. (Albany Med. Coll., N.Y.). *Bull. Environ. Contam. Toxicol.* 13(4):424-432, 1975.

5478 INFLUENCE OF CYTOPLASMIC FACTORS ON PROTEIN SYNTHESIS IN RAT LIVER. *(E.)* Von Der Decken, A. (Wenner-Gren Inst. Exp. Biol., Stockholm, Sweden). *Acta Biol. Med. Ger.* 33(5-6):839-843, 1975.

5479 INFLUENCE OF PHENOBARBITONE ON THE SIZE AND ACTIVITY FOR PROTEIN SYNTHESIS OF LIVER POLYRIBOSOMES FROM FASTED RATS. *(E.)* Ragnotti, G.; Aletti, M. G. (Inst. Gen. Pathol., Milan, Italy). *Acta. Biol. Med. Ger.* 33(5-6):855-858, 1974.

5480 EFFECT OF PHENOBARBITAL AND CARBON TETRACHLORIDE ON SODIUM DEHYDROCHOLATE METABOLISM IN THE GUINEA-PIG. *(E.)* Chenderovitch, J.; Raizman, A.; Infante, R. (INSERM, Hosp. Saint-Antoine, Paris, France). *Biol. Gastroenterol. (Paris)* 63(7): 265-274, 1974.

5481 SULFATED GLYCOPROTEINS IN NORMAL HUMAN BILE. *(E.)* Nagashima, H.; Suzuki, N.; Yosizawa, Z. (Tohoku Univ. Sch. Med., Sendai, Japan). *Tohoku J. Exp. Med.* 113(3):225-229, 1974.

5482 FOLIC ACID POLYGLUTAMATE CONJUGASE ACTIVITY IN LIVER OF RATS RECEIVING DIPHENYLHYDANTOIN. *(E.)* Honda, Y.; Arakawa, T. (Tohoku Univ. Sch. Med., Sendai, Japan). *Tohoku J. Exp. Med.* 113(2):197-199, 1974.

5483 INDUCTION OF CHOLANGIOCARCINOMA FOLLOWING TREATMENT OF A RHESUS MONKEY WITH AFLATOXIN. *(E.)* Tilak, T. B. G. (Dep. Patho., Nat. Inst. Nutr., Hyderabad, India). *Food Cosmet. Toxicol.* 13(2):247-249, 1975.

5484 ADRENERGIC RECEPTORS IN RAT LIVER II. EFFECTS OF PHENYLEPHRINE, ISOPROTERENOL AND ADRENERGIC BLOCKING AGENTS ON GLUCONEOGENESIS IN PERFUSED LIVER. *(E.)* Hagino, Y.; Nakashima, M. (Nagoya Univ. Sch. Med., Japan). *Jpn. J. Pharmacol.* 24(3):373-381, 1974.

5485 HISTOPATHOLOGICAL STUDIES OF THE EFFECTS OF CYTOCHROME c ON CARBON TETRACHLORIDE-INDUCED LIVER DAMAGE AND ON THE LIVERS OF AGED RATS. *(E.)* Ohnishi, H.; Hayashi, Y.; Ogawa, N.; Yajima, G.; Aihara, K. (Nippon Med. Coll., Tokyo, Japan). *Jpn. J. Pharmacol.* 24(3):425-432, 1974.

5486 THE EFFECT OF THYROIDECTOMY ON THE SUBCELLULAR Mg DISTRIBUTION IN RAT LIVER. *(E.)* Magalhaes, M. C. D. (Lab. Pharmacol., Inst. Gulbenkian Ciencia, Oeiras, Portugal). *Life Sci.* 16(7):1089-1094, 1975.

5487 INTERFERENCE OF ETHANOL IN HEPATIC CELLULAR METABOLISM. *(E.)* Lieber, C. S. (VA Hosp., Bronx, New York). *Ann. N.Y. Acad. Sci.* 252: 24-50, 1975.

5488 PROTEIN SYNTHESIS IN THE HEPATOCYTE. *(E.)* Oratz, M.; Rothschild, M. A.; Schreiber, S. (VA Hosp., New York, N.Y.). *Ann. N.Y. Acad. Sci.* 252:57-62, 1975.

5489 INTERFERENCE OF ETHANOL IN CELLULAR METABOLISM. *(E.)* Lundquist, F. (Dep. Biochem., Univ. Copenhagen, Denmark). *Ann. N.Y. Acad. Sci.* 252:11-20, 1975.

5490 ALTERATIONS IN HEPATIC CHROMATIN TEMPLATE AVAILABILITY DURING INFECTION. *(E.)* Earp, H. S. (U.S. Army Med. Res. Inst. Inf. Dis., Fort Detrick, Md.). *Am. J. Physiol.* 228(4):1183-1187, 1975.

5491 TOXICOLOGICAL IMPLICATIONS OF THE MIXED-FUNCTION OXIDASE CATALYZED METABOLISM OF CARBON DISULFIDE. *(E.)* Dalvi, R. R.; Hunter, A. L.; Neal, R. A. (Vanderbilt Univ. Sch. Med., Nashville, Tenn.). *Chem.-Biol. Interact.* 10(5):347-361, 1975.

5492 EVALUATION OF PERFUSED LIVER VIABILITY. *(E.)* Ruwart, M. J.; Kaminski, D. L.; Jellinek, M. (St. Louis Univ., Mo.). *Fed. Proc.* 34(3):395, 1975.

5493 DISTRIBUTION OF NEWLY FORMED FATTY ACIDS AMONG GLYCEROLIPIDS OF ISOLATED PERFUSED RAT LIVER. *(E.)* Kuksis, A.; Myher, J. J.; Marai, L.; Yeung, S. K. F.; Steiman, I.; Mookerjea, S. (Banting Best Dep. Med. Res., Toronto, Canada). *Can. J. Biochem.* 53(5):509-578, 1975.

5494 DISTRIBUTION OF DIPEPTIDASE ACTIVITY BETWEEN LYSOSOMES AND SOLUBLE FRACTION OF RAT LIVER. *(E.)* Taylor, S. L.; Tappel, A. L. (Dep. Food Sci. Technol., Univ. California, Davis). *Can. J. Biochem.* 53(5):502-508, 1975.

5495 NUCLEAR PROTEIN-KINASE ACTIVITY IN PERFUSED RAT LIVER STIMULATED WITH DIBUTYRYL-ADENOSINE CYCLIC 3':5'-MONOPHOSPHATE. *(E.)* Castagna, M.; Palmer, W. K.; Walsh, D. A. (Sch. Med., Univ. California, Davis). *Eur. J. Biochem.* 55(1):193-199, 1975.

5496 HEPATIC CARBOHYDRATE METABOLIC ALTERATIONS DURING ENDOTOXIC AND TRAUMATIC SHOCK. *(E.)* Filkins, J. P.; Buchanan, B. J.; Cornell, R. P. (Stritch Sch. Med., Maywood, Ill.). *Circ. Shock* 2(2):129-135, 1975.

5497 THE EFFECT OF DIETARY VITAMIN E ON GLUTATHIONE-INDUCED LIVER MITOCHONDRIAL SWELLING FROM MICE TREATED WITH 3'-4-METHYL-4-DIMETHYLAMINOAZOBENZENE. *(E.)* Block, J. A.; Roy, R. M. (Dep. Biol. Sci., Sir George Williams Univ., Montreal, Canada). *Experientia* 31(5):537-538, 1975.

5498 ORTHOTOPIC LIVER TRANSPLANTATION UTILIZING A VASCULAR STAPLING INSTRUMENT. *(E.)* Barron, P. T.; Vogelfanger, I. J.; Waddell, W. G. (Dep. Surg., Univ. Ottawa, Ontario, Canada). *Br. J. Surg.* 62(6):438-440, 1975.

5499 INDUCTION OF HEPATITIS IN ADULT SYRIAN HAMSTERS BY H-1 VIRUS. *(E.)* Henry, C. J.; Diorio, A. F. (Allegheny General Hosp., Pittsburgh, Pa.). *Proc. Soc. Exp. Biol. Med.* 149(1):23-28, 1975.

5500 EFFECT OF CARBON TETRACHLORIDE ON BILIARY EXCRETION OF CERTAIN FOREIGN ORGANIC COMPOUNDS IN THE RAT. *(E.)* Barakat, M. Z.; Abou-El-Makarem, M. M. (Dep. Biochem., Azhar Univ., Cairo, Egypt). *Bull. Acad. Pol. Sci. (Biol.)* 22(7-8):465-469, 1974.

5501 AN ELECTRON MICROSCOPIC STUDY OF THE HEPATOCYTES IN EXPERIMENTALLY INDUCED SHOCK CONDITIONS. *(E.)* Ohba, H.; Aihara, K.; Masugi, Y.; Yajima, G. (Nippon Med. Sch. Tokyo, Japan). *J. Clin. Electron Microsc. (Tokyo)* 7(3-4):363-364, 1974.

5502 ULTRASTRUCTURAL CHANGES OF THE LIVER AND KIDNEYS AND LUNGS IN EXPERIMENTALLY INDUCED THERMAL BURNS. *(E.)* Joong, S. T.; Youn, K. H.; Yajima, G.; Asano, G.; Hirayama, T.; Hayashi, M.; Kuno, Y.; Arai, K.; Tooyama, M.; Miyazaki, H. (Dep. Pathol., Kyunpook Nat. Univ., Japan). *J. Clin. Electron. Microsc. (Tokyo)* 7(3-4):365-366, 1974.

5503 ULTRASTRUCTURAL STUDIES ON EXPERIMENTAL PHOSPHOLIPIDOSIS AFTER GALACTOSAMINE INDUCED HEPATIC INJURY IN RAT LIVER CELLS. *(E.)* Takino, T.; Sugino, S.; Sugihara, T.; Okanoue, T.; Horii, Y.; Masuda, M. (Kyoto Prefectural Univ. Med., Japan). *J. Clin. Electron Microsc. (Tokyo)* 7(3-4): 372-373, 1974.

5504 LONG-TERM STRUCTURAL ALTERATIONS OF THE MOUSE LIVER THROUGH DL-α-TOCOPHEROL OVERDOSE. *(E.)* Kurono, C.; Watari, N.; Koyama, K. (Nagoya City Univ. Med. Sch., Japan). *J. Clin. Electron Microsc. (Tokyo)* 7(3-4):379, 1974.

5505 THE EFFECT OF ASPIRIN (ACETYL SALICYLATE) ON MACROMOLECULE TURNOVER IN RAT KIDNEY AND LIVER. *(E.)* Wheldrake, J. F. (Sch. Biol. Sci., Univ. South Australia, Bedord). *Experientia* 31(5): 559-560, 1975.

5506 HEPATIC RADIATION INJURY: EARLY AND LATE. *(E.)* Kinzie, J. J.; Swartz, H.; Hensley, G.; Griem, M. L. (Dep. Radiol., Univ. Chicago, Ill.). *Radiat. Res.* 62(3):543, 1975.

5507 HEPATIC LEVELS OF CYCLIC AMP IN NORMAL AND LEAD-SENSITIZED RATS AFTER TREATMENT WITH BACTERIAL ENDOTOXIN. *(E.)* Gartner, S. L. (Naval Med. Res. Inst., Bethesda, Md.). *Experientia* 31(5): 566-567, 1975.

5508 INFLUENCE OF PARTIAL HEPATECTOMY ON LATE STRUCTURAL CHANGES IN THE LIVER FOLLOWING THE LOCAL IRRADIATION. *(E.)* Jovanovic, D. A.; Bemelmans, D. H. (Inst. Canc. Kapucijnenvoer, Leuven, Belgium). *Radiat. Res.* 62(3):543, 1975.

5509 HEPATIC FUNCTION AND VASCULAR ALTERATION FOLLOWING PARTIAL LIVER X-IRRADIATION IN THE RAT. *(E.)* Jackson, K. L.; Christensen, G. M. (Dep. Radiol., Univ. Washington, Seattle). *Radiat. Res.* 62(3):543-544, 1975.

5510 THE DEVELOPMENT OF A NEW METHOD OF HEPATECTOMY BY RADIATION OF MICROWAVES. *(E.)* Osaka, A. (Sapporo City Hosp., Japan). *Bull. Soc. Int. Chir.* 33(3):166-171, 1974.

5511 STIMULATION OF LIVER REGENERATION AND COMPENSATORY KIDNEY HYPERPLASIA BY PASSIVE TRANSFER OF SPLEEN CELLS. *(E.)* Pliskin, M. E.; Prehn, R. T. (Fox Chase Cancer Cent., Philadelphia, Pa.). *J. Reticuloendoendothel. Soc.* 17(5):290-299, 1975.

5512 RELEASE OF LYSOSOMAL ENZYME β-GLUCURONIDASE AFTER HEPATIC CRYOSURGERY. *(E.)* Nagasue, N. (Kyushu Univ. Sch. Med., Fukuoka, Japan). *Arch. Surg.* 110(6):713-716, 1975.

5513 ELECTRONOMICROSCOPIC MORPHOMETRIC CHANGES IN THE LIVER CELL OF RATS FOLLOWING ADMINISTRATION OF ALCOHOL AND ALCOHOL-RESERPIN. *(E.)* Dragiev, M.; Hristozova, A.; Mitkov, D. (I. P. Pavlov Higher Med. Inst., Plovdiv, Bulgaria). *Folia Med. (Plovdiv)* 16(2):83-93, 1974.

5514 ALLOTRANSPLANTATION IN STUDIES OF HEPATIC PHYSIOLOGY. *(E.)* Ranson, J. H. C.; Eng, K.; Rapaport, F. T.; Localio, S. A. (New York Univ. Sch. Med., N.Y.). *Transplant. Proc.* 7(1):759-762, 1975.

5515 DESCRIPTION OF THE ANHEPATIC STATE AND ACUTE HEPATIC FAILURE: EXPERIMENTAL STUDY IN PIGS. *(E.)* Galmarini, D.; Tarenzi, L.; Vercesi, G.; Beffagna, B.; Cantaluppi, G.; Mantovani, A.; Pellegrini, S.; Giradello, D.; Vitali, B. (Surg. Clin., Univ. Milan, Italy). *Surg. Ital.* 4(2):102-117, 1974.

5516 LIPIDS IN THE LIVER OF SQUIRREL, AFTER CARBON TETRACHLORIDE POISONING. *(E.)* Rana, S. V. S. (Dep. Zoology, D. A. V. Coll., Muzaffarnagar, India). *Acta Histochem.* 51(2):214-219, 1974.

5517 TECHNIQUE FOR ASSESSMENT OF LOCAL EFFECTS OF SUBSTANCES FOUND IN BILE UPON OPENING PRESSURE OF CHOLEDOCHODUODENAL JUNCTION. *(E.)* Tansey, M. F.; Innes, D. L.; Martin, J. S.; Kendall, F. M. (Temple Univ. Sch. Dent. Pharm., Philadelphia, Pa.). *J. Pharm. Sci.* 64(7):1174-1177, 1975.

5518 ULTRASTRUCTURAL EVIDENCE OF THE HEPATOTOXIC EFFECT OF HALOTHANE IN RATS FOLLOWING IN-UTERO EXPOSURE. *(E.)* Chang, L. W.; Lee, Y. K.; Dudley, Jr., A. W.; Katz, J. (Dep. Pathol. Anesthesiol., Univ. Wisconsin, Madison). *Can. Anaesth. Soc. J.* 22(3):330-338, 1975.

5519 THE RATE OF DEGRADATION OF LIVER PROTEINS. *(E.)* Kim, Y. S.; Choi, B. H.; Kim, Y. (St. Louis Univ. Sch. Med., Mo.). *Exp. Mol. Pathol.* 22(3):342-349, 1975.

5520 ON THE PROBLEM OF A COMMON HEPATIC TRANSPORT PROCESS FOR STERIODS. UPTAKE OF ^{3}H-OUBAIN, ^{3}H-TAUROCHOLIC ACID AND ^{3}H-CORTICOSTERONE INTO RAT LIVER SLICES. *(E.)* Erttmann, R. R.; Damm, K. H. (Dep. Pharmacol., Univ. Hamburg, W. Germany). *Arch. Int. Pharmacodyn.* 214(2):232-239, 1975.

5521 HISTOLOGICAL EFFECTS OF LIPIDS ON THE LIVER AND SPLEEN OF MICE. *(E.)* Stuart, A. E.; Smith, I. I. (Dep. Pathol., Univ. Edinburgh, Scotland). *J. Pathol.* 115(2):63-71, 1974.

5522 UTILIZATION OF AMINO ACIDS BY THE ISOLATED PERFUSED SHEEP LIVER. *(E.)* Lindsay, D. B.; Jarrett, I. G.; Mangan, J. L.; Linzell, J. L. (A.R.C. Inst. Anim. Physiol., Babraham, England). *Q. J. Exp. Psychol.* 60(2):141-149, 1975.

5523 EFFECT ON GUINEA-PIGS OF FEEDING NITROSOMORPHOLINE AND ITS PRECURSORS IN COMBINATION WITH ASCORBIC ACID. *(E.)* Akin, F. J.; Wasserman, A. E. (U.S. Dep. Agric., Athens, Ga.). *Food Cosmet. Toxicol.* 13(2):239-242, 1975.

5524 AFLATOXIN B_1 HYDROXYLATION BY HEPATIC MICROSOMAL PREPARATIONS FROM THE RHESUS MONKEY. *(E.)* Krieger, R. I.; Salhab, A. S.; Dalezios, J. I.; Hsieh, D. P. H. (Dep. Environ. Toxicol., Univ. Calif., Davis). *Food Cosmet. Toxicol.* 13(2):211-219, 1975.

5525 RIBOFLAVIN AND MOUSE HEPATIC CELL STRUCTURE AND FUNCTION. MITOCHONDRIAL OXIDATIVE METABOLISM IN SEVERE DEFICIENCY STATES. *(E.)* Hoppel, C. L.; Tandler, B. (Dep. Pharmacol., Case Western Reserve Univ., Cleveland, Ohio). *J. Nutr.* 105(5):562-570, 1975.

5526 INTERACTIONS OF CADMIUM WITH COPPER, IRON, ZINC AND MANGANESE IN OVINE TISSUES. *(E.)* Doyle, J. J.; Pfander, W. H. (Anim. Sci. Res. Cent., Univ. Mo., Columbia). *J. Nutr.* 105(5):599-606, 1975.

5527 FREE RADICAL EVOLUTION IN RAT LIVER FOLLOWING THE INGESTION OF DAB. *(E.)* Duchesne, J.; Goutier, R.; Marechal, R.; Van De Vorst, A. (Dep. Atomic Mol. Phys., Univ. Liege, Belgium). *Phys. Med. Biol.* 20(2):305-309, 1975.

5528 CARBOXYMETHYL HORSE-LIVER ALCOHOL DEHYDROGENASE. LIGAND-BINDING AND KINETIC PROPERTIES OF THE CYSTEINE-46-MODIFIED ENZYME. *(E.)* Reynolds, C. H.; McKinley-McKee, J. S. (Univ. Edinburgh Med. Sch., Scotland). *Arch. Biochem. Biophys.* 168(1):145-162, 1975.

5529 REGULATION OF RAT LIVER β-HYDROXY-β-METHYLGLUTARYL COENZYME A REDUCTASE ACTIVITY AND CHOLESTEROL LEVELS OF SERUM AND LIVER IN VARIOUS DIETARY AND HORMONAL STATES. *(E.)* Lakshmanan, M. R.; Dugan, R. E.; Nepokroeff, C. M.; Ness, G. C.; Porter, J. W. (VA Hosp., Madison, Wis.). *Arch. Biochem. Biophys.* 168(1):89-95, 1975.

5530 METABOLISM OF CHLOROBENZENE WITH HEPATIC MICROSOMES AND SOLUBILIZED CYTOCHROME P-450 SYSTEMS. *(E.)* Selander, H. G.; Jerina, D. M.; Daly, J. W. (Nat. Inst. Arthritis, Metab., Dig. Dis., Bethesda, Md.). *Arch. Biochem. Biophys.* 168(1):309-321, 1975.

5531 EFFECT OF CLOFIBRATE ON THE ADENOSINE TRIPHOSPHATASE ACTIVITY OF RAT LIVER MITOCHONDRIA. *(E.)* Panini, S. R.; Kurup, C. K. R. (Dep. Biochem., Indian Inst. Sci., Bangalore, India). *Arch. Biochem. Biophys.* 168(1):188-197, 1975.

5532 ROLE OF LIVER FUNCTIONS ON LIVER CELL MITOSIS. *(E.)* Takata, T. (Okayama Univ. Med. Sch., Japan). *Acta Med. Okayama* 28(3):199-212, 1974.

5533 VASOPRESSIN AND HEPATIC ARTERY. EFFECT OF SELECTIVE CELIAC INFUSION OF VASOPRESSIN ON THE HEPATIC ARTERY FLOW. *(E.)* Barr, J. W.; Lakin, R. C.; Rosch, J. (Univ. Oregon Med. Sch., Portland). *Invest. Radiol.* 10(3):200-205, 1975.

5534 INCREASE IN SERUM ALPHA FOETOPROTEIN LEVEL IN HEPATIC REGENERATION OF THE RAT. EFFECTS OF AGE AND OF MAGNITUDE OF REGENERATIVE ACTIVITY. *(E.)* Nayak, N. C.; Mital, I.; Dhar, A.; Chopra, P.; Das, P. K. (All India Inst. Med. Sci., New Delhi). *Br. J. Exp. Pathol.* 56(2):56, 113-118, 1975.

5535 DIVERSE MECHANISMS OF HEPATOCELLULAR INJURIES DUE TO CHEMICALS: EVIDENCE IN RATS ADMINISTERED CARBON TETRACHLORIDE OR DIMETHYLNITROSAMINE. *(E.)* Nayak, N. C.; Chopra, P.; Dhar, A.; Das, P. K. (All India Inst. Med. Sci., New Delhi). *Br. J. Exp. Pathol.* 56(2):56, 103-112, 1975.

5536 CHOLESTEROL FATTY LIVER: MORPHOLOGICAL CHANGES IN THE COURSE OF ITS DEVELOPMENT IN RABBITS. *(E.)* Lee, S. S.; Ho, K. J. (Univ. Alabama Med. Cent., Birmingham). *Arch. Pathol.* 99(6): 301-306, 1975.

5537 PROTECTION OF THE ISCHEMIC LIVER BY DONOR PRETREATMENT BEFORE TRANSPLANTATION. *(E.)* Toledo-Pereyra, L. H.; Simmons, R. L.; Najarian, J. S. (Dep. Surg., Univ. Minnesota, Minneapolis). *Am. J. Surg.* 129(5):513-517, 1975.

5538 SYMPOSIUM: NUTRITIONAL PERSPECTIVES AND ATHEROSCLEROSIS. NONNUTRITIVE FIBER AND LIPID METABOLISM. *(E.)* Kritchevsky, D.; Tepper, S. A.; Story, J. A. (Wistar Inst. Anat. Biol., Philadelphia, Pa.). *J. Food Sci.* 40(1):8-11, 1975.

5539 INHIBITION OF LIVER ALDEHYDE DEHYDROGENASE BY PYROGALLOL AND RELATED COMPOUNDS. *(E.)* Rubenstein, J. A.; Collins, M. A.; Tabakoff, B. (Dep. Biochem. Biophys., Loyola Univ., Chicago, Ill.). *Experientia* 31(4):414-415, 1975.

5540 DISTRIBUTION OF NEWLY FORMED PALMITATE AND STEARATE AMONG MOLECULAR SPECIES OF CHOLINE AND ETHANOLAMINE PHOSPHATIDES. *(E.)* Kuksis, A.; Myher, J. J.; Marai, L.; Yeung, S. K. F.; Steiman, I.; Mookerjea, S. (Banting Best Dep. Med. Res., Toronto, Canada). *Can. J. Biochem.* 53(5):519-526, 1975.

5541 EXPERIMENTAL STUDIES ON THE INFLUENCE OF THIAMAZOL ON CLINICOCHEMICAL PARAMETERS OF CHOLESTASIS. *(Ger.)* Obwald, V. H.; Hadhanyi, A.; Heidenreich, O. (Med. Clin., Technical Univ., Munich, Germany). *Arzneim. Forsch.* 24(12):1992-1995, 1974.

5542 PINOCYTOSIS AND DEGRADATION OF FOREIGN PROTEINS IN LIVER PARENCHYMAL CELLS. EXPERIMENTAL STUDY IN THE MOUSE LIVER. *(Ger.)* Arnold, W.; Muller, O.; Von Mayerbach, H. (Med. Clin. II, Johannes Gutenberg Univ., Mainz, Germany). *Z. Gastroenterol.* 13(3):404-406, 1975.

5543 THE EFFECT OF PROLONGED INGESTION OF MINERAL OIL ON THE PIG: SELECTIVE RETENTION AND INTERFERENCE WITH CHOLESTEROL METABOLISM. *(Fr.)* Tulliez, J.; Bories, G.; Peleran, J. C. (Res. Lab. Aliment. Addit., Natl. Inst. Agron. Res., Toulouse, France). *C. R. Acad. Sci. [D] (Paris)* 280(19):2261-2264, 1975.

5544 EXPERIMENTAL STUDIES WITH ANIMALS ON GALLSTONE FORMATION, PROPHYLAXIS AND REGRESSION IN VITAMIN C DEFICIENCY AND APPLICATION OF PREDNISOLONE. *(Ger.)* Schubert, S.; Grosser, L.; Fuchs, K. F.; Wohlgemuth, B.; Bellman, H. (Surg. Clin., Karl Marx Univ., Leipzig, Germany). *Z. Exp. Chir.* 8(1): 41-48, 1975.

5545 TURNOVER OF NEUTRAL LIPIDS AND PHOSPHOLIPIDS IN RAT LIVER MITOCHONDRIA. *(It.)* Megli, F. M.; Landriscina, C.; Quagliariello, E. (Inst. Biol. Chem., Univ. Bari, Italy). *Boll. Soc. Ital. Biol. Sper.* 50(17):1342-1348, 1974.

5546 THE EFFECT OF CYCLIC AMP ON FATTY ACID SYNTHESIS IN SUBCELLULAR FRACTIONS OF RAT LIVER. *(It.)* Groni, G. V.; Landriscina, C.; Quagliariello, E. (Inst. Biol. Chem., Univ. Bari, Italy). *Boll. Soc. Ital. Biol. Sper.* 50(17):1335-1341, 1974.

5547 POSSIBILITIES OFFERED BY NEW MOLECULES IN HEPATIC FUNCTIONAL EXPLORATION. *(Fr.)* Meyniel, M. G.; Lapalus, F.; Moreau, M. F. (INSERM, Paris, France). *Bordeaux Med.* 8(6):605-611, 1975.

5548 PHARMACOLOGICAL ASPECTS OF S-ADENOSYLMETHIONINE IN HEPATOLOGY. *(It.)* Stramentinoli, G.; Pezzoli, C.; Catto, E. (BioRes. Liscate, Milan, Italy). *Minerva Med.* 66(33):1541-1562, 1975.

5549 SECRETION OF HEPATIC TRIGLYCERIDES INTO PLASMA OF RATS FED RETINOL. *(E.)* Ahuja, H. C.; Misra, U. K. (Vallabhbhai Patel Chest Inst., Univ. Delhi, India). *Agr. Biol. Chem.* 39(3):637-644, 1975.

5550 EFFECTS OF PHENOBARBITAL ON PROTEIN SYNTHESIS AND POLYSOME LEVELS IN RAT LIVER. *(E.)* Venkatesan, N.; Steele, W. J. (Univ. Iowa, Coll. Med., Iowa City). *Chem. Biol. Interact.* 11(1):41-54, 1975.

5551 OXIDATIVE DESATURATION OF α-LINOLENIC, LINOLEIC, AND STEARIC ACIDS BY HUMAN LIVER MICROSOMES. *(E.)* De Gomez Dumm, I. N. T.; Brenner, R. R. (Inst. Physiol., Univ. Nat., La Plata, Argentina). *Lipids* 10(6):315-317, 1975.

5552 IDENTIFICATION AND QUANTITATION OF CHOLANOIC ACIDS IN HEPATIC AND EXTRA-HEPATIC TISSUES OF RAT. *(E.)* Oh, S. Y.; Dupont, J. (Dep. Food Sci. Nutr., Colorado State Univ., Fort Collins). *Lipids* 10(6):340-347, 1975.

5553 ACTION OF *n*-ALKANES ON DRUG-METABOLIZING ENZYMES FROM GUINEA-PIG LIVER. *(E.)* Notten, W. R. F.; Henderson, P. T. (Inst. Pharmacol., Univ. Nijmegen, Netherlands). *Biochem. Pharmacol.* 24(10):1093-1097, 1975.

5554 THE METABOLISM OF FOREIGN COMPOUNDS IN RATS AFTER TREATMENT WITH POLYCHOLORINATED BIPHENYLS (PCBs). *(E.)* Grote, W.; Schmold, A.; Dammann, H. G. (Dep. Pharmacol., Univ. Hamburg, Germany). *Biochem. Pharmacol.* 24(10):1121-1125, 1975.

5555 CYCLIC ADENOSINE 3':5'-MONOPHOSPHATE AND THE INDUCTION OF DEOXYRIBONUCLEIC ACID SYNTHESIS IN LIVER. *(E.)* Short, J.; Tsukada, K.; Rudert, W. A.; Lieberman, I. (VA Hosp., Pittsburgh, Pa.). *J. Biol. Chem.* 250(10):3602-3606, 1975.

5556 INCREASED LEVELS OF RAT HEPATIC NUCLEAR FREE AND ENGAGED RNA POLYMERASE ACTIVITIES DURING LIVER REGENERATION. *(E.)* Yu, F. L. (Jefferson Med. Coll., Philadelphia, Pa.). *Biochem. Biophys. Res. Commun.* 64(3):1107-1116, 1975.

5557 MECHANISM FOR THE INHIBITION OF β-OXIDATION BY ETHANOL: THE ROLE OF THE MALATE-ASPARTATE SHUTTLE. *(E.)* Lumeng, L.; Bremer, J.; Davis, E. J. (Indiana Univ. Sch. Med., Indianapolis, Inc.). *Gastroenterology* 68(4):938, 1975.

5558 TETRAHYDROFURANE--AN INHIBITOR FOR ETHANOL-INDUCED LIVER MICROSOMAL CYTOCHROME P450. *(E.)* Ullrich, V.; Weber, P.; Wollenberg, P. (Dep. Physiol. Chem., Univ. Saarland, Homburg, W. Germany). *Biochem. Biophys. Res. Commun.* 64(3):808-813, 1975.

5559 ULTRASTRUCTURAL EVOLUTION OF EXPERIMENTALLY INDUCED LIPID INCLUSIONS IN RAT AND MOUSE HEPATOCYTES INDICATIVE FOR THE RECOVERY OF LIVERS FROM EFFECTS OF BENZIDAMINE AND TRIACETYLOLEANDOMYCIN. *(E.)* Garaci, E.; Djaczenko, W. (Inst. Microbiol., Univ. Rome, Italy). *Ann. Sclavo.* 16(1):18-28, 1974.

5560 INHIBITORY EFFECT OF PROLACTIN ON THE DEVELOPMENT OF FATTY LIVER INDUCED BY ACTH IN THE RAT. *(E.)* Ozegovic, B.; Milkovic, S. (Lab. Exp. Med., Univ. Zagreb., Yugoslavia). *Proc. Soc. Exp. Biol. Med.* 149(1):262-265, 1975.

See also, 5210, 5218, 5294, 5297, 6096, 6133, 6153, 6194, 6345.

SECRETION AND METABOLISM

Intestine

5561 ENZYME-HISTOCHEMICAL AND ELECTRON MICROSCOPE STUDIES OF THE CIRCADIAN RHYTHM IN RAT INTESTINAL MUCOSA. *(Ger.)* Schafer, V. A.; Hohn, P.; Mika, H.; Allbach, G. (Inst. Pathol., Johannes Gutenburg Univ., Mainz, Germany). *Acta Histochem. (Jena)* 48(2):301-319, 1974.

Circadian rhythms of the small intestine were studied in Sprague-Dawley rats which were screened from outside disturbances. The animals were divided into six groups of seven rats each, and sections of the bowel were removed at six fixed points at the following times of the day: 20, 24, 4, 8, 12, and 16 hr. The bowel sections were removed from locations ranging from the duodenum, jejunum and ileum to the start of the ascending colon. Enzyme functions were evaluated by electron microscopy and histochemistry (alkaline phosphatase, acid phosphatase, glucose-6-phosphatase, nucleotidase, ATP, leucinaminopeptidase, LDH, succinodehydrogenase, monoaminooxidase, and glucose-6-phosphatedehydrogenase). The hydrolases in particular showed clear-cut fluctuations of activity. In most cases the values in the afternoon and night hours were significantly higher than in the early morning and day time hours. The findings with the dehydrogenases were less uniform. G-6PDH and MAO showed relatively small differences of activity at various times of the day, whereas SDH showed a maximum at 8 hr and LDH a peak at 8 hr and 24 hr. The data presented

are in agreement with the concept of the existence of circadian fluctuations of activity in the intestinal mucosa which in a general sense are an expression of the nocturnal activity of the rat.

5562 LYSOSOMAL CHANGES IN THE GUT OF RATS AFTER CHRONIC TREATMENT WITH PHENOBARBITAL. *(E.)* Rivera-Calimlim, L.; Bosmann, H. B.; Penney, D. P.; Lasagna, L. (Univ. Rochester Sch. Med. Dent., N.Y.). *Biochem. Pharmacol.* 24(11-12):1161-1166, 1975.

The effect of long-term oral phenobarbital administration on the lysosomal enzyme activity of the intestine was studied in male Sprague-Dawley rats. Phenobarbital in distilled water solution (100 mg/kg/day) was administered by intragastric tube for 8 weeks. Matched controls were treated with distilled water. The intestinal transport of ^{14}C-methionine and ^{14}C-D-xylose in everted jejunal sacs, the ultrastructure of the jejunum, and the lysosomal enzyme activity of the jejunum were subsequently studied. Significant increases in the lysosomal enzyme activities in the intestine were observed 1 and 2 months after the beginning of phenobarbital treatment. As early as 1 month after the commencement of treatment, the activities of α-glucosidase, β-galactosidase, β-*N*-acetyl-galactosaminidase, β-*N*-acetyl-glucosaminidase, β-xylosidase, and acid phosphatase were increased, as were cathepsin-like and trypsin-like activities; the activities of α-mannosidase and α-fucosidase increased only after 2 months of treatment. Ultrastructural changes in the jejunum of the drug-treated rats were also observed; these included increased agranular endoplasmic reticulum and ribosomal particles and increases in the acid-phosphatase-positive dense bodies and coated vesicles in the absorptive cells of the intestine. There were no gross morphological changes in the intestine and there were no major alterations in the morphology of the goblet cells or in the mitochondria, terminal web, microvillus projections, Golgi apparatus, or nuclei of the absorptive cells. In addition, the mucosal transport of ^{14}C-methionine and ^{14}C-D-xylose was not altered. Thus, chronic administration of phenobarbital causes biochemical and morphological changes in the intestine; this dose level corresponds to a dose of about 600 mg in man, which is within the daily dose range administered to human patients.

5563 REGIONAL AND CELLULAR LOCALIZATION OF GLYCOSYLTRANSFERASES IN RAT SMALL INTESTINE: CHANGES IN ENZYMES WITH DIFFERENTIATION OF INTESTINAL EPITHELIAL CELLS. *(E.)* Kim, Y. S.; Perdomo, J.; Ochoa, P.; Isaacs, R. A. (Veteran's Admin. Hosp., San Francisco, Calif.). *Biochim. Biophys. Acta* 391(1):39-50, 1975.

Optimal assay conditions were determined for four glycosyltransferases in rat small intestinal mucosal homogenates and the regional distribution and cellular localization of these enzymes was studied. Samples of jejunum and ileum were prepared from male Wistar rats, homogenized and incubated with a mixture containing Fetuin II or Fetuin I acceptor, buffer, $MnCl_2$, ATP, UDPglucose, UDP(U-^{14}C)galactose or CMP-N-acetyl(4,5,6,7,8,9-^{14}C)-neuraminic acid, Triton X-100, and enzyme. For each glycosyltransferase, similar levels of activity were found in duodenal, proximal jejunal, and distal ileal segments; the activities of the galactosyltransferases being lower in the distal jejunal-proximal ileal segments. Planar section studies indicated that undifferentiated crypt cells had significantly higher levels of sialyltransferase activities of the jejunum and ileum than the mature villous cells. A similar crypt-to-villous gradient was found for a galactosyltransferase in the ileum. Requirements for the galactosyltransferase assay included Mn^{2+}, Triton X-100, UDPglucose, and acceptor, while the sialyltransferase activity required Triton X-100 and acceptor. With Fetuin II as acceptor, the optimal conditions for the assay of the two galactosyltransferases were pH 6.0, 45 C, and 10 mM Mn^{2+}; with ovine submaxillary mucin as acceptor, the optimum assay conditions were pH 7.0, 37 C, and 40 mM $MnCl_2$. For the sialyltransferase assays, pH 6.1 and 30 C were optimal. The results suggest that the glycoprotein synthesis may be active on the undifferentiated crypt cells and that certain glycosyltransferases may serve as marker enzymes for cellular differentiation in the intestine.

5564 OCCURRENCE OF H_2-RECEPTORS FOR HISTAMINE IN THE GUINEA-PIG INTESTINE. *(E.)* Bareicha, I.; Rocha E Silva, M. (Fac. Med., Sao Paulo, Brazil). *Biochem. Pharmacol.* 24(11-12):1215-1219, 1975.

The specific anti H_2 agent, burimamide, was used to analyze the stimulatory effects of histamine on the isolated guinea-pig ileum, and the proportion of H_2/H_1 receptors was calculated by the application of Clark's equation to data obtained in the presence and absence of burimamide. Isolated strips of guinea-pig ileum and duodenum were perfused with medium containing 10^{-7} M atropine; dose-response curves were obtained by adding (in triplicate or quadruplicate) increasing doses of histamine in the absence (control) and presence of 10^{-6} M burimamide. Burimamide potentiated the stimulatory effect of histamine on the guinea-pig ileum, an effect which was attributed to the relaxing effect induced by H_2-receptors present in the preparation. Similar results were obtained with pieces of guinea-pig duodenum. On the basis of Clark's equation, the proportion of H_2/H_1 receptors in the ileum was calculated to be 15.7%, while the average value for the duodenum was 12.4%. This small percentage of H_2/H_1 receptors may account for the fact that only the smallest concentrations of histamine appeared to be potentiated by burimamide and that this effect tended to disappear as the concentrations of histamine approached those giving maximal contraction.

5565 STUDIES ON THE BRUSH BORDER MEMBRANE OF THE MOUSE DUODENUM. I. MEMBRANE ISOLATION AND ANALYSIS OF PROTEIN COMPONENTS. *(E.)* Billington, T.; Nayudu, P. R. V. (Dep. Zool., Monash Univ., Victoria, Australia). *J. Membr. Biol.* 21(1-2):49-64, 1975.

The proteins, carbohydrates and lipids of the brush border membrane of the mouse duodenum were analyzed using duodenal segments from adult Swiss mice. Brush border membranes were isolated from the villus epithelial cells of the segments, solubilized in Tris-sodium dodecyl sulfate buffer, and analyzed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. Preparations of these membranes were not contaminated by other organelles as judged from electron micrographs of sectioned brush-border pellets. Electrophoresis of the brush border proteins revealed a reproducible Coomassie Brilliant Blue pattern of 17 bands. By comparing the brush border protein band positions with those of standard proteins run concurrently on sodium dodecyl sulfate-polyacrylamide gel slabs, it was estimated that the 17 proteins and subunits had molecular weights ranging from over 250,000 to around 16,000 daltons. Periodate-fuchsin sulfite staining showed that the five slowly migrating, high molecular weight proteins were glycoproteins. The two proteins of smallest molecular size reacted positively with Oil Red O but had very small amounts of lipophilic amino acid residues, indicating that the lipid extractable from the gels in these areas was a contaminant and not bound to the proteins.

5566 EFFECT OF STASIS ON INTESTINAL ENZYME ACTIVITIES. *(E.)* Gracey, M.; Thomas, J.; Houghton, M. (Princess Margaret Child. Med. Res. Found., Perth, Western Australia). *Aust. N.Z. J. Med.* 5(2):141-144, 1975.

The intestinal mucosal activities of lactase, sucrase, maltase, alkaline phosphatase, and N-acetyl-β-glucosaminidase were studied in rats with surgically-induced upper intestinal stasis. Upper jejunal blind loops were made in adult Wistar rats. After 4-8 weeks tissue specimens were taken from the blind loop, the jejunum 5 cm proximal to the junction with the loop, and the jejunum 5 cm distal to the loop; specimens were also taken from the duodenojejunal junction in normal control rats. Activities of the three disaccharidases were significantly reduced in all three anatomical regions studied, but the reduction was more marked in the blind loop specimens than in those from the proximal or distal jejunum. Activities of alkaline phosphatase and N-acetyl-β-glucosaminidase were also significantly reduced in the distal gut and blind loop tissues; in addition, alkaline phosphatase was significantly reduced in the proximal gut. The results suggest a causal relation between the degree of overgrowth of the small intestinal microflora, the extent of bile salt deconjugation, and the degree of mucosal damage in the blind-loop animals; they also suggest that there was a considerable degree of damage to the lysosymes in this experimental model. The pathogenetic significance of these findings in relation to the "contaminated small-bowel syndrome" in man is unclear, but they suggest that there is an important and previously ignored mucosal lesion in this syndrome.

5567 LOCALIZATION OF SUBSTANCE P-LIKE IMMUNOREACTIVITY IN MOUSE GUT. *(E.)* Nilsson, G.; Larsson, L. I.; Hakanson, R.; Brodin, E.; Pernow, B.; Sundler, F. (Dep. Pharmacol., Karolinska Inst., Stockholm, Sweden). *Histochemistry* 43(1):97-99, 1975.

Specific antibodies against synthetic substance P were used to localize this peptide in the mouse intestine. Sectioned duodenal and proximal colonic specimens from female mice were treated with substance P antiserum and examined under the fluorescence microscope using the indirect immunofluorescence method. Strong immunofluorescence was observed in numerous nerve fibers of the myenteric plexus in the colon but not in the duodenum. The immunofluorescent nerve fibers encircled the ganglionic cells, which were nonfluorescent, and extended from the plexus in between bundles of smooth muscle. Immunofluorescence was also observed in scattered epithelial cells in the mucosa of both colon and duodenum, their numbers being much greater in the latter than the former. The immunoreactivity of these endocrine-like cells was of lower intensity than that of the nerve fibers of the myenteric plexus. In number, shape, and distribution, the immunoreactive mucosal cells resembled the enterochromaffin cells. Thus, substance P-like immunoreactivity occurs in the myenteric plexus of the proximal colon and in epithelial, possible endocrine, cells of the mucosa.

5568 LOCALIZATION OF THE MONOGLYCERIDE PATHWAY ENZYMES IN THE VILLUS TIPS OF INTESTINAL CELLS AND THEIR ABSENCE FROM THE BRUSH-BORDER. *(E.)* Negrel, R.; Ailhaud, G. (Cent. Biochim., Univ. Nice, France). *FEBS Lett.* 54(2):183-188, 1975.

The localization of monoglyceride pathway enzymes within the intestinal epithelial cells of the rat and pig was studied. Intestinal epithelial cells were fractionated, and purified brush-border vesicles were prepared. Aminopeptidase, alkaline phosphatase, cytochrome c oxidase, NADPH-cytochrome c reductase, Acyl-coenzyme A (CoA):monoglyceride and diglyceride acyltransferases, Na^+, K^+-ATPase ouabain sensitive activity, inorganic P, and N-acetylglucosaminyltransferase were assayed. Acyl-Coa:monoglyceride acyltransferase was localized in the villus tips of both the rat and pig intestinal cells. The acyl-CoA: diglyceride acyltransferase showed a similar pattern, with a ratio of mono:diglyceride acyltransferase equal to 1:3. Sucrose density gradient analysis and precipitation of the brush-border membrane vesicles by aminopeptidase antibodies indicated that the acyl-CoA:monoglyceride acyltransferase was not present in the brush-border fraction. The small amount of enzyme activity responsible for the monoglyceride esterification in the brush-border fraction is due to contamination by membranous structures. The results demonstrate that the acyl-CoA:monoglyceride acyltransferase (as well as the acyl-Coa:diglyceride acyltransferase), the specific marker of the monoglyceride pathway, can be used as a marker of differentiation for intestinal cells. The membranous structures to which the monoglyceride acyltransferase is attached may be some smooth endoplasmic reticulum in continuity with the cytoplasmic membrane.

5569 EFFECT OF ACUTE DIETARY ALTERATION UPON INTESTINAL LIPID SYNTHESIS. *(E.)* Mansbach, C. M., II. (VA Hosp., Durham, N.C.). *Lipids* 10(6):318-321, 1975.

The specific activities of 3 enzymes engaged in complex lipid synthesis, diglyceride acyltransferase, cholinephosphotransferase, and lysolecithin acyltransferase, were studied in intestinal mucosa of hamsters fed 2 meals of either saline, hydrolyzed casein, or corn oil in a 5.5-hr period. The animals were sacrificed 4 hr later and the small intestine divided into 4 equal segments; the mucosa of each segment was scraped to yield villous tip and crypt preparations, from which low-speed supernatant and microsomal fractions were derived. Enzyme assays, thin layer chromatography, and protein and radioactivity determinations were made. In the most proximal intestine, saline feeding was associated with reduced specific activities of all 3 enzymes in villous tips when compared with the 2 calorie-supplemented groups. In the most distal intestine, oil feeding increased the activity of lysolecithin acyltransferase and cholinephosphotransferase 2-fold as compared to casein-fed hamsters; diglyceride acyltransferase was increased 1.5-fold. The response of lysolecithin acyltransferase and diglyceride acyltransferase to fat feeding was incomplete when compared to hamsters fed a fat-supplemented diet for 7 days, suggesting that their pattern of response to fat was similar to sucrase response to sucrose feeding. By contrast, the response of cholinephosphotransferase to fat feeding was complete at 24 hr, suggesting that it responds in a manner similar to the glycolytic enzymes.

5570 THE "FAT RAT": A NEW EXPERIMENTAL JEJUNOILEAL BYPASS MODEL. *(E.)* Madura, J. A.; Usicsko, J. F.; Cooney, D. R.; Grosfeld, J. L. (Indiana Univ. Sch. Med., Indianapolis). *J. Pediatr. Surg.* 10(3):349-352, 1975.

Morphologic and metabolic alterations induced by jejunoileal bypass were studied in a unique obese experimental animal referred to as the "fat rat". A 90% jejunoileal bypass was performed in 20 genetically obese Zucker fat rats (500-600 g) and 20 Sprague-Dawley rats (350-400 g). Twenty unoperated growing rats of each strain served as controls. All of the animals were evaluated daily for weight loss, postoperative complications, and survival. At the end of 40 days, the animals were sacrificed and liver sections were evaluated microscopically. All rats with a jejunoileal bypass lost weight; the most rapid loss occurred during the 1st postoperative week. At 40 days, the mean weight loss for the 19 surviving operated Sprague-Dawley rats was 50 g; the controls had a mean weight gain of 35 g. The 16 surviving operated fat rats had a weight loss of 175 g while the 20 controls had a gain of 80 g. Minimal fatty infiltration of the liver was seen in 1 of 20 Sprague-Dawley controls; minimal to moderate fatty infiltration was observed in all fat rat controls. Moderate fatty infiltration was observed in the operated Sprague-Dawley rats, but operated fat rats consistently demonstrated moderate to severe fatty infiltration of the liver. The data suggest that the fat rat is a valuable laboratory model for studying morbid obesity and the aftereffects of jejunoileal bypass.

5571 EFFECTS OF PHARMACOLOGIC SYMPATHECTOMY ON GUT HYPERTROPHY IN ENTERECTOMIZED RATS. *(E.)* Touloukian, R. J.; Tilson, M. D.; Roth, R. (Yale Univ. Sch. Med., New Haven, Conn.). *J. Surg. Res.* 19(1):9-11, 1975.

The effects of 6-hydroxydopamine-induced sympathectomy on the compensatory hypertrophy seen in the jejunal and ileal remnants following enterectomy were studied in rats to further define the relation between adrenergic innervation and gut hypertrophy. 6-Hydroxydopamine (75 mg/kg) was injected i.v. into male Sprague-Dawley rats; the injection was repeated after 7 days. One week after the second injection, the sympathectomized animals and a group of normal animals underwent 50% mid-enterectomy. The jejunal and ileal remnants were examined 2 months later and the villus length measured. An 80% reduction in the catecholamine concentration of the jejunum and ileum was observed 1 week and 1-2 months after 6-hydroxydopamine treatment. This treatment had no effect on the growth rate of the animals or villus height of the jejunal or ileal remnants after enterectomy. These data do not support the hypothesis that adrenergic innervation initiates or accelerates hypertrophy in the gut remnant.

5572 EFFECT OF BILE ACIDS ON ELECTRICAL PROPERTIES OF RAT COLON: EVALUATION OF AN IN-VITRO MODEL FOR SECRETION. *(E.)* Schwiter, E. J.; Hepner, G. W.; Rose, R. C. (Pennsylvania State Univ. Coll. Med., Hershey). *Gut* 16(6):477-481, 1975.

Changes in the short-circuit current and transmural electrical potential difference (pd) of the rat colon were measured as an index of the state of secretory activity of the tissue following bile acid administration. The serosa and part of the musculature were removed from proximal colonic segments taken from male Sprague-Dawley rats. The remaining tissue was incubated in Ringer solution containing bile acid (2 mM) and/or theophylline (10 mM). The short-circuit current and pd were measured before and after the addition of bile acid and theophylline. Applied to the serosal and mucosal surfaces or to the serosal surface alone, taurochenodeoxycholic acid increased the short-circuit current and pd within 2 min; this acid was without effect when applied to the mucosal surface alone. Taurochenodeoxycholic acid also produced a significant decrease in tissue resistance. When this acid was added to the mucosal and/or serosal solution after the addition of theophylline, there was no increase in short-circuit current. Taurocholic acid also increased short-circuit current and pd when applied to serosal and mucosal surfaces or the serosal surface alone; however, the taurocholic acid-induced increase in the short-circuit current following sero-

sal application was significantly less than that seen with taurochenodeoxycholic acid. Theophylline increased the short-circuit current and pd when applied to the serosal and/or mucosal surface. Theophylline had no significant effect on either parameter when added after the serosal application of taurochenodeoxycholic acid, although it had a stimulatory effect when added after the mucosal application of this acid; pretreatment with taurocholic acid at either surface slightly reduced the effect of theophylline. In that these results are inconsistent with results obtained *in vivo*, it is concluded that this *in vitro* model has limitations for studying the mechanism of bile acid-stimulated intestinal secretion.

5573 EFFECTS OF DIETS CONTAINING DRIED WHEY ON THE LACTASE ACTIVITY OF THE SMALL INTESTINAL MUCOSA AND THE CONTENTS OF THE SMALL INTESTINE AND CECUM OF THE PIG. *(E.)* Ekstrom, K. E.; Benevenga, N. J.; Grummer, R. H. (Coll. Agricultural Life Sciences, Univ. Wisconsin, Madison, Wis. 53706). *J. Nutr.* 105(7):851-860, 1975.

The effects of long-term feeding of various levels of dried whey on the mucosal lactase-specific activities of several genetic groups of pigs were studied. Additional experiments investigated the effect of feeding a high level of lactose on the total lactase activities of intestinal mucosa and contents, as well as the distributional pattern of these activities along the intestinal tract of two breeds of swine. Feeding 0, 10, 25 or 40% dried whey in the first study did not alter the mucosal lactase-specific activity. Assay of duodenal lactase, for example, showed 0.16 μM of galactose released/mg of protein/hr in controls, compared to values of 0.22, 0.18 and 0.18, resp., for the 10%, 25% and 40% whey diets, resp. There was no significant difference between breeding groups or breed-treatment interactions. The types of breeds used were not specified. In the second study, lactase activity in the small intestine was estimated in the total activity found in 10-cm segments of each meter of intestine. Feeding a diet containing 40% whey continuously from weaning (age 5 weeks) or from age 12 weeks did not alter the total lactase activity in pigs killed at age 21 weeks. Dietary lactose tended to increase lactase activity in cecal and small intestinal contents, but the increases were not always statistically significant. Chester White pigs had approximately 3-fold greater mucosal lactase activities (815 g of lactose able to be hydrolyzed in 24 hr) than those of Hampshire pigs (268 g). It is suggested that these two breeds may be suitable models to study the relations between lactase deficiency and lactose intolerance. Further, these results indicate that the level of mucosal lactase activity of the pig small intestine is not altered by dietary lactose.

5574 RELEASE OF CHOLECYSTOKININ BY ACID. *(E.)* Barbezat, G. O.; Grossman, M. I. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Proc. Soc. Exp. Biol. Med.* 148(2):463-467, 1975.

The amount of cholecystokinin (CCK) released by acid was estimated *in vivo* in mongrel dogs prepared with chronic pancreatic fistulas and Thomas cannulas in the dependent portion of the stomach. Porcine secretin (1300 ng/kg/hr) was given by continuous i.v. infusion and the pancreatic secretion was allowed to reach a steady level before the infusion of HCl (or 0.15 M NaCl to controls) and phenol red into the duodenum. In some cases, additional secretin or 10% porcine CCK was given in place of the duodenal infusion. In other experiments, the pancreas was stimulated for 2 hr by: continuous duodenal perfusion with 0.16 M HCl at 12.5, 25, 50, 100, or 200 ml/hr; continuous i.v. infusion of synthetic secretin at 62.5, 125, 250, or 500 ng/kg-hr; or continuous i.v. infusion of a fixed ratio combination of synthetic secretin and CCK. The volume, bicarbonate, and protein concentrations of the pancreatic juice were measured. Duodenal infusion of 16 nM/hr of HCl with a high background dose of i.v. secretin stimulated pancreatic protein secretion to about the same extent as did 0.5 u/kg-hr of exogenous CCK. The same acid load without the secretin background gave a response equivalent to about 0.7 u/kg-hr of CCK. Thus, expressed as fractions of their respective doses for half maximal response, acid releases almost five times more secretin than CCK.

5575 PROPERTIES OF GUANYLATE CYCLASE AND LEVELS OF CYCLIC GMP IN RAT SMALL INTESTINAL VILLOUS AND CRYPT CELLS. *(E.)* De Jonge, H. R. (Fac. Med., Erasmus Univ. Rotterdam, The Netherlands). *FEBS Lett.* 55(1):143-152, 1975.

The characteristics of brush border-localized guanylate cyclase were compared with those of microsomal enzyme preparations from isolated villous and crypt cells. A variety of hormones and hormone-like substances reported to raise cyclic AMP levels in the rat small intestine, to elevate cyclic GMP in other tissues, or to affect intestinal transport functions were tested for their ability to alter the intestinal guanylate cyclase activity *in vitro* or to regulate cyclic GMP levels in isolated villous and crypt cells and in mucosal scrapings. The kinetic properties of the brush border-bound guanylate cyclase included a positive co-operative behavior indicating multiple binding sites for $MnGTP^{2-}$ and independence from free Mn^{2+} at saturating $MnGTP^{2-}$ concentrations. The kinetic patterns of the particulate gunaylate cyclase from the upper villous cell microsomes, on the other hand, showed a marked loss of co-operativity with respect to $MnGTP^{2-}$ and activation by free Mn^{2+} at high $MnGTP^{2-}$ levels. Guanylate cyclase from the crypt microsomes displayed an intermediate type of kinetic behavior. Incubation with the brushborder guanylate in the presence of 0.1% Triton X-100 gave a 2-fold rise in activity, while 1.4 mM of $CaCl_2$ slightly inhibited the enzyme at 0.3 mM Mn^{2+}free but stimulated it by 1.2-fold at 4 mM Mn^{2+}free. Strong inhibitory effects were found with PEP, oxaloacetate, and ATP or ADP. Alloxan had a slightly inhibitory effect, amino acids in physiological concentrations had a small stimulatory effect, and D-isomers were ineffective, as were NaF, glucose, choleragen, prostaglandins E_1, F_2 and A_1, concanavalin A, and a number of catecholamines, cholinergic agents, and

peptide hormones. The latter agents were also ineffective in stimulating the enzyme from the villous and crypt cell homogenates, microsomes, and mucosal scrapings. The basal levels of cyclic GMP decreased from crypt to villous cells, while the guanylate cyclase activity increased. $CaCl_2$ slightly depressed the cyclic GMP levels, while alloxan strongly elevated the cyclic GMP levels but had no effect on the cyclic AMP levels. Theophylline significantly elevated the cyclic AMP levels but did not affect the cyclic GMP levels. Ionophore A-23187, choleratoxin, prostaglandins, concanavalin A, and various cholinergic agents and hormones had no effect on the cyclic GMP levels.

5576 EFFECTS OF FASTING AND FEEDING AND PILOCARPINE ON PANETH CELLS OF THE MOUSE. *(E.)* Ahonen, A.; Penttila, A. (Dep. Anat., Univ. Helsinki, Finland). *Scand. J. Gastroenterol.* 10(4):347-352, 1975.

The effects of pilocarpine and fasting, with or without refeeding, on the Paneth cells of the mouse were studied. Fasted adult mice of both sexes were treated i.p. with nialamide (500 mg/kg), followed 1.5 hr later with pilocarpine (100 mg/kg, i.p.), and 0.5 hr after that with dopamine (100 mg/kg, i.p.); controls were treated only with nialamide and dopamine. Another group of mice was fasted for 2 days, then, in some cases, allowed to eat for 2 hr, and then treated with nialamide and dopamine as above. The animals were killed 1, 2, 3, 4, 5, or 6 hr after dopamine administration and samples of the duodenum and midjejunum were stained and examined by light and fluorescence microscopy; the dopamine-labeled secretory granules of the Paneth cells were counted. Pilocarpine stimulated the extrusion of granules from the cytoplasm of the cells and significantly decreased the number of granules, whereas fasting for 2 days without refeeding significantly increased the number of granules/cell. Refeeding following 2 days fasting had no effect on the Paneth cells of the duodenum, but resulted in a significant decrease in the number of granules in the jejunal Paneth cells compared with the number seen after fasting without refeeding. The data indicate that pilocarpine is able to stimulate the secretory mechanism of the Paneth cells, and suggest that the secretory mechanisms of these cells may be regulated in a different way in various regions of the gastrointestinal tract.

5577 RESULTS OF ORTHOTOPIC HOMOTRANSPLANTATION OF THE SMALL BOWEL IN THE SWINE. *(Fr.)* Hay, J. M.; Fagniez, P. L.; Parc, R.; Kunlin, A.; Villiers, P.; Vaiman, M.; Remy, J.; Bsowsky, A.; Chomette, G.; Clot, J. P.; Nizza, P.; Garnier, H. (Appl. Radiobiol. Lab., Commis. At. Energy, JOUY-en-JOSUS, France). *Ann. Chir.* 28(12):1063-1070, 1974.

5578 THE EFFECT OF RADIOPROTECTIVE BIOGENIC AMINES ON LIPID PEROXIDATION OF MICROSOMES OF RAT INTESTINAL MUCOSA. *(Rus.)* Sobolev, A. S.; Merzliak, M. N.; Zhamsaranova, S. D.; Goncharenko, E. N.; Kudriashov, Iu. B. (M. V. Lomonosov Moscow State Univ., USSR). *Dokl. Akad. Nauk SSSR* 222(2):476-479, 1975.

5579 EVIDENCE OF MAJOR ROLE OF THE INTESTINE IN CHOLESTEROL SYNTHESIS IN THE ADULT MALE RAT. *(E.)* Chevallier, F.; Magot, T. (Lab. Physiol. Nutr., Univ. Paris, France). *Experientia* 31(6):627-629, 1975.

5580 GLUCOSE-STIMULATED REVERSAL OF SECRETION TO ABSORPTION IN INTESTINAL OBSTRUCTION. *(E.)* Coan, M. G.; Costantini, F.; Fellner, J.; Wright, H. K. (Yale Univ. Sch. Med., New Haven, Conn.). *Surg. Forum* 24:420-421, 1973.

5581 TIME/DOSE RELATIONSHIPS IN ABDOMINAL IRRADIATION: A DEFINITION OF PRINCIPLES AND EXPERIMENTAL EVALUATION. *(E.)* Hagemann, R. F.; Concannon, J. P. (Allegheny Gen. Hosp., Pittsburgh, Pa.). *Br. J. Radiol.* 48(571):545-555, 1975.

5582 ELECTROFOCUSING SEPARATION OF INTESTINAL MALTASES IN THE RAT. *(E.)* Suduka, P.; Delmont, J.; Rigat, A. M. (Fac. Med., Nice, France). *Digestion* 11(5/6):459-460, 1974.

5583 ON THE SPECIFICITY OF THE ENZYME RELEASE IN THE SMALL INTESTINE OF THE RAT. *(E.)* Roduner, J.; Honegger, J.; Hauri, H. P.; Goetze, H.; Hadorn, B. (Dep. Pediatr., Univ. Berne, Switzerland). *Digestion* 11(5/6):457-458, 1974.

5584 MUSCARINIC RECEPTORS IN RAT INTESTINAL MUSCLE: COMPARISON WITH THE GUINEA PIG. *(E.)* Taylor, I. K.; Cuthbert, A. W.; Young, M. (Univ. Cambridge, Med. Sch., England). *Eur. J. Pharmacol.* 31(2):319-326, 1975.

5585 PREDICTION OF INTESTINAL VIABILITY USING DOPPLER ULTRASOUND TECHNICS. *(E.)* Wright, C. B.; Hobson, II, R. W. (Walter Reed Army Med. Cent., Washington, D. C.). *Am. J. Surg.* 129(6):642-645, 1975.

5586 STUDIES OF THE EFFECTS OF BLOCKAGE OF INTESTINAL LYMPHATICS: III. TEMPORARY ISOLATION OF LYMPHEDEMATOUS BOWEL FROM INTESTINAL CONTINUITY. *(E.)* Wilk, P.; Karipineni, R.; Dreiling, D. A.; Danese, C. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *Am. J. Gastroenterol.* 63(5):400-403, 1975.

5587 DETECTION OF CIRCULATING ANTIGEN IN RATS WITH SMALL BOWEL ADENOCARCINOMA. *(E.)* Englund, C. W.; Stevens, R. H.; Cheng, H. F.; Osborne, J. W. (Med. Lab., Iowa Univ., Iowa City). *Radiat. Res.* 62(3):603, 1975.

5588 RADIATION-INDUCED INTESTINAL TUMORS IN RATS: CORRELATION BETWEEN THE AMOUNT OF THE CIRCULATING TUMOR-RELATED ANTIGEN(S) AND TUMOR SIZES. *(E.)* Cheng, H. F.; Cantarero, L.; Osborne, J. W. (Med. Lab., Univ. Iowa, Iowa City). *Radiat. Res.* 62(3):604, 1975.

5589 5-FLUOROURACIL MODIFICATION OF POST-IRRADIATION CELL PROLIFERATION IN THE MOUSE JEJUNUM. *(E.)* Burholt, D. R.; Copper, J. W.; Hagemann, R. F.; Schenken, L. L.; Lesher, S. (Allegheny Gen. Hosp., Pittsburgh, Pa.). *Radiat. Res.* 62(3):545, 1975.

See also, 5199, 5200, 5203, 5204, 5221, 5224, 5226, 5240, 5244, 5276, 5296, 5425, 5601, 5602, 5607, 5610.

GENERAL

5590 EFFECT OF ASPIRIN, BILE SALT, AND ETHANOL ON CANINE GASTRIC MUCOSAL BLOOD FLOW. *(E.)* Cheung, L. Y.; Moody, F. G.; Reese, R. S. (Univ. Utah Coll. Med., Salt Lake City). *Surgery* 77(6): 786-792, 1975.

The effect of aspirin (20 mM), sodium taurocholate (40 mM), and ethanol (25%) on gastric mucosal blood flow was studied in an exteriorized, chambered preparation of canine fundic stomach. Gastric mucosal blood flow was measured by gamma-labeled microspheres and plasma aminopyrine clearance. All three test solutions caused a significant increase in microsphere-measured mucosal blood flow which was proportional to the severity of gross mucosal injury, being greatest with bile salt and least with ethanol. Plasma aminopyrine clearance determined only a small fraction of mucosal flow measured by microspheres following injury, particularly in sodium taurocholate and ethanol experiments, where the net hydrogen ion back diffusion was greatest. The results suggest that increased gastric mucosal blood flow may be a secondary defensive response to the damage caused by these irritants and that plasma aminopyrine clearance is unreliable in quantitating mucosal blood flow changes in the presence of mucosal injury.

5591 STRUCTURAL AND HORMONAL ALTERATIONS IN THE GASTROINTESTINAL TRACT OF PARENTERALLY FED RATS. *(E.)* Johnson, L. R.; Copeland, E. M.; Dudrick, S. J.; Lichtenberger, L. M.; Castro, G. A. (Univ. Texas Med. Sch., Houston). *Gastroenterology* 68(5, Part 1):1177-1183, 1975.

The structural and hormonal properties of the gastrointestinal tract were compared in six male Sprague-Dawley rats maintained by i.v. hyperalimentation and in six sham-operated controls. Control rats gained twice as much weight (78 *versus* 34 g) as parenterally-fed animals over a 19-day period. Kidney, spleen and testes weights did not differ between the two groups. Weights of a 2 cm portion of small intestine and of whole stomach were 73% and 25% greater, resp., in the control rats. Although antral weights were unchanged, antral gastrin was decreased by 30-fold in parenterally-fed animals. Oxyntic gland area and forestomach weights were decreased by 35%. The decrease in antral gastrin occurred early within the first 8 days after initiation of hyperalimentation. Total small intestine DNA content was decreased 2-fold and the RNA:DNA ratio was increased almost 2-fold in parenterally-fed rats, suggesting gut hypoplasia with hypertrophy of remaining cells in the experimental group. Pancreatic weight was decreased by 67% in hyperalimented rats. These results indicate that oral food ingestion is necessary to maintain structural integrity of certain tissues and accessory glands of the gastrointestinal tract and to maintain normal tissue gastrin stores.

5592 FISSION OF CRYPTS IN THE SMALL INTESTINE OF THE IRRADIATED MOUSE. *(E.)* Cairnie, A. B.; Millen, B. H. (Dep. Biol., Queens Univ., Kingston, Canada). *Cell Tissue Kinet.* 8(2):189-196, 1975.

Mouse small intestine was irradiated to determine if the number of crypts is eventually restored to normal after irradiation, and to determine the mechanism of increase in crypt number. LAF1 mice were given whole body irradiation at 21 or 66 rads/min. Tritiated thymidine (100 μCi) was injected i.p. 1 hr before sacrifice. Three mice each were killed at 3, 5, 7, 14, and 21 days after irradiation at 21 rads/min. There were also three unirradiated controls. Mice given 66 rads/min were killed: three at 2, 3, 4, and 5 days; two at 6, 7, 14 and 21 days; three at 32, and two unirradiated controls at 32 days. Small pieces of epithelium were dissected from three widely separated areas of the intestine, the villi and muscular layer removed and the crypts counted. They were then placed in scintillation vials for determination of tritium activity. The number of villi in fragments from the same three areas were counted and the tritium activity determined. Crypt and villus counts were only made for the mice exposed to the lesser dose. At 1 day after irradiation, crypts were collapsed in appearance. At 3 days there were a few obvious regenerating crypts, and by 5 days there were enough regenerating crypts to begin counting (0.27×10^6 crypts, as compared to 1.18×10^6 in the controls). The number steadily increased until it approached normal at 21 days, but the crypts were larger than normal. Very large crypts were found at 4-7 days, and budding was observed on the sides and bases of many. Forked crypts, suggestive of fission, were seen from 6 days on. These were not seen before 5 days, or in controls. It is concluded that increase in crypt number is at least partially due to fission of existing crypts, and that there is probably a compensatory feedback control regulating the number of crypts/animal.

5593 CULTURE OF ZOLLINGER-ELLISON TUMOR CELLS. *(E.)* Lichtenberger, L. M.; Lechago, J.; Dockray, G. J.; Passaro, Jr., E. (Univ. California Los Angeles Sch. Med.). *Gastroenterology* 68(5):1119-1126, 1975.

Gastrin-secreting Zollinger-Ellison tumor cells were grown *in vitro* to allow study of the molecular form of the hormone, the kinetics of cell growth and the effects of stimulants and inhibitors on cell growth. Tumor biopsies were obtained from six patients, and incubated in a minimal essential medium at 37 C. The medium was replaced every third day, and the used medium stored and radioimmunoassayed for gastrin. Cells were subcultured onto coverslips, fixed, and stained with hematoxylin and eosin. For gastrin radioimmunoassay, pure, natural G-17 gastrin was used as both standard and label (on iodination with ^{125}I). Used medium was incubated with trypsin and fractionated by Sephadex chromotography. To calculate doubling time, a 1:100 cell dilution was seeded; after cells became adherent, various fields containing single cells were marked, left for a week, and the cells/resulting clone were counted, after 7, 21, 49, and 70 hr. For electron microscopy, cells were fixed in glutaraldehyde, and postfixed and stained with osmium tetroxide. To measure gastrin concentration after stimulation, old medium was replaced with 5.0 ml fresh fetal calf medium, and 0.25 ml samples were removed at 2.5, 10, 20 and 30 min. In all cases, explants adhered to the glass and cell outgrowths occurred within a week. Cell population consisted of epithelial and fibroblast-like cells in varying ratios, the latter becoming predominant after 2-3 months. Cells increased in number exponentially, and doubling time was 65 hr. Cultures derived from tumors at the head of the pancreas or adjacent lymph nodes had the highest tissue and medium hormone levels, while the lowest levels were from large liver tumors. In all cases, medium gastrin concentration decreased with time; there were negligible amounts after 2-7 weeks. In about 5% of the cultures, gastrin was still present after 2 months. Gel filtration chromatography of culture medium showed a major peak of radioimmunoassayable gastrin before the ^{125}I-gastrin standard, with an elution volume closely corresponding to that of G-34. There was also a minor peak of gastrin eluting at the expected location of unlabeled G-17 I. Fractionation of tumor extract showed a major peak of G-17 and a minor one of G-34, the reverse of the cultured cells. Medium gastrin concentration increased 4-10 fold for 30 min after addition of fresh medium in two lines. Over 60% of the cells from a line cultured for 2 weeks contained cytoplasmic granules 150-200 nm in diameter, in contrast to the 250-300 nm diameter granules in the original tumor. Prominent rough endoplasmic reticulum and Golgi bodies were present in both. After 6 weeks in culture, less than 10% of the cells contained a few granules, while lysosomes and autophagic vesicles were more common. It is concluded that cell doubling time was 65 hr, gastrin secretion decreased with time as epithelial cells were replaced by fibroblasts, and fresh medium stimulated secretion.

5594 FUNCTIONAL STUDIES OF PEYER'S PATCHES: EVIDENCE FOR THEIR PARTICIPATION IN INTESTINAL IMMUNE RESPONSES. *(E.)* Muller-Schoop, J. W.; Good, R. A. (Sloan-Kettering Inst. Cancer Res., New York, N.Y.). *J. Immunol.* 114(6):1757-1760, 1975.

The pattern of responsiveness of Peyer's patch, splenic, mesenteric, and inguinal node lymphocytes was studied in guinea pigs following p.o. and parenteral antigenic stimulation. Live bacille Calmette Guerin (BCG) bacilli or heat-killed mycobacteria (*Mycobacterium tuberculosis*) were administered to male Hartley guinea pigs *via* stomach tube; in other animals, heat-killed mycobacteria in Freund's adjuvant were injected into the left front footpad. The guinea pigs were exsanguinated and lymphocyte suspensions prepared from the intestinal Peyer's patches, spleens, and mesenteric and inguinal lymph nodes. These cultures were stimulated *in vitro* with purified tuberculin derivative (PPD) or with Clostridia. After p.o. immunization with BCG, the Peyer's patch lymphocytes showed the best *in vitro* proliferative responses to PPD. Responsiveness to Clostridia was also significant in the Peyer's patch lymphocytes and low or absent in the other lymphoid tissues examined. In animals immunized parenterally with heat-killed mycobacteria, the splenic lymphocytes reacted best to PPD, the Peyer's patch lymphocytes giving the smallest reaction. Selective elimination of B or T cells from the PPD-reactive Peyer's patch lymphocyte populations showed that the T cell-rich fraction remained responsive to PPD, while the B cell-rich fraction became unresponsive; both fractions reacted to Clostridia. The results indicate that a functional T cell population is present in the Peyer's patch and that these organs in the guinea pig are immunocompetent lymphoid organs which seem to be primarily engaged in the production of immune responses to antigens presented from the intestinal lumen.

5595 THE DIGESTIVE TRACT IN TOTAL PARENTERAL NUTRITION. *(E.)* Koga, Y.; Ikeda, K.; Inokuchi, K.; Watanabe, H.; Hashimoto, N. (Kyushu Univ. Sch. Med., Fukuoka, Japan). *Arch. Surg.* 110(6):742-745, 1975.

The effects of long-term complete parenteral nutrition on the morphology of different parts of the digestive tract were studied in 8- to 10-week-old puppies. The puppies were maintained for 4 or 8 weeks on an i.v. solution providing 140 calories/kg/day; control animals were fed a commercial diet providing the same number of calories/day. The animals were killed on the last day of the experiment and measurements were made of the length and weight of the small intestine. The thickness of the mucosa and muscular coat of the small intestine and the number of cells in mitosis and the number of argentaffin cells among the epithelial cells of the jejunal crypts were also measured. In the puppies maintained on total parenteral nutrition, the development of the gastrointestinal tract was markedly disturbed, with reductions in length, weight, and thickness of all intestinal layers; in general, these changes were more marked in the 8-week experimental group. In contrast, the microscopic structures were well preserved. There was no noticeable difference between the experimental and control animals in gastric pit:gland ratio, villus:crypt ratio, cellular component, content of periodic acid-Schiff-positive substance, or the distribution of sudanophilic fat in the mucosal membrane. However, there was a reduction in the mitotic index in the

jejunal crypt in the experimental animals. Growth, development, and weight gain were basically normal in the 4-week experimental animals, but weight gain was somewhat reduced in the 8-week group. Stool output of the experimental animals was scanty, and mucus stools were evacuated once or twice/week. The results suggest that in complete parenteral nutrition, cell production in the digestive tract may be decreased due to decreased cell loss. They also suggest that the passage of diet through the small intestine is essential for the development of length in this organ.

5596 IMMUNOGLOBULIN E (IgE) AND IgE-CONTAINING CELLS IN HUMAN GASTROINTESTINAL FLUIDS AND TISSUES. *(E.)* Brown, W. R.; Borthistle, B.K.; Chen, S. T. (Dep. Med., Denver VA Hosp., Colo.). *Clin. Exp. Immunol.* 20(2):227-237, 1975.

The distribution of IgE immunocytes along the intestine and the IgE content of the intestinal fluids were studied in healthy human subjects and in patients with various digestive diseases. Samples of gastric, duodenal, jejunal, rectal, and colonic tissues were examined with immunohistological and single- and double-antibody radioimmunoassay techniques; the intestinal fluid samples were also examined by single- and double-antibody radioimmunoassay and the 4 specimens containing the largest quantities of IgE were analyzed by sucrose gradient ultracentrifugation. IgE-containing cells were identified in all tissue specimens examined, although they were less numerous than IgA, IgG, and IgM immunocytes; they comprised about 2% of the total immunofluorescent-positive cell population. The total number of immunoglobulin-forming cells of all classes, the absolute number of IgE-containing cells, and the percentage of IgE-containing cells decreased along the intestine from stomach to rectum. IgE immunocytes were at least 10-fold more numerous at the bases of gastric and duodenal peptic ulcers than elsewhere in these tissues, and the number of IgE-type cells in the rectal tissues of 2 patients with Crohn's disease and 1 patient with chronic ulcerative colitis was 3- to 5-fold greater than in the rectal tissues of subjects with other or no colonic diseases. Measurable IgE was found in 92% of the intestinal fluid samples; the concentration did not differ significantly among normal subjects and patients with various digestive diseases. Sucrose gradient ultracentrifugation analysis revealed that the immunologically reactive IgE was largely present in fractions corresponding to molecules of lower molecular weight than that of albumin, which suggest that IgE in the gut contents is degraded by proteolytic enzymes. The results suggest that IgE antibodies are available for participation in local reaginic-type reactions in the gut.

5597 ESCAPE FROM VASOCONSTRICTION IN THE GASTRIC MICROCIRCULATION. *(E.)* Guth, P. H.; Smith, E. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Am. J. Physiol.* 228(6):1893-1895, 1975.

The possible occurrence of escape from vasoconstriction in the gastric microcirculation, and the involvement of submucosal shunt opening in this phenomenon, was studied in male Wistar rats. Using an image-splitting TV microscope recording system, the response of the gastric submucosal arterioles (13-33 μm) to 3 min of left splanchnic nerve stimulation (8 V), norepinephrine superfusion (0.2-0.7 μg/min), and vasopressin superfusion (.0025-.005 U/min) was studied. All stimuli produced initial vasoconstriction, the average maximum vasoconstriction diameter being 72-79.9% of control values. With sympathetic nerve stimulation and norepinephrine superfusion, the initial vasoconstriction was followed by escape in all animals, the average escape index (relative degree of escape from vasoconstrictor stimulation) being 0.64 after sympathetic nerve stimulation and 0.77 after norepinephrine superfusion. In neither case was significant poststimulation dilatation observed. In contrast, escape occurred in only 1 animal after vasopressin superfusion, the mean escape index in this group being -0.50; again, significant poststimulation dilatation was not observed. No arteriovenous shunts were seen. The results indicate that the gastric submucosal arterioles exhibit an escape phenomenon following adrenergic stimulation, suggesting that "autoregulatory escape" in other splanchnic beds may also be due to the relaxation of constricted vessels and not to the opening of shunts.

5598 VITAMIN B_{12} ABSORPTION IN X-IRRADIATED RATS. *(E.)* D'Souza, D. W.; Vakil, U. K.; Sreenivasan, A. (Biochem. Food Technol. Div., Bhabha At. Res. Cent., Bombay, India). *Environ. Physiol. Biochem.* 5(1):37-48, 1975.

The effect of whole-body exposure to a sublethal dose of x-irradiation on the intestinal submucosal absorption of vitamin B_{12} was studied in male Wistar rats. Weanling rats were provided with adequate nutrition or maintained on vitamin A-deficient diets; after 3 weeks, the animals were placed on low (8%) or adequate (20%) diets. Two weeks later, the animals were x-irradiated (400 rad) or were sham-irradiated. Vitamin B_{12} absorption was measured in intestinal loop segments following the injection of vitamin B_{12} (with or without gastric juice from normal rats) into several ligated intestinal segments. The distribution of radioactivity in the serum and tissues was also measured after i.p. (0.5 μCi) or p.o. (1 μCi) administration of ^{57}Co-B_{12}. The rate of absorption of vitamin B_{12} from the intestinal loops of adequately nourished rats was decreased by x-irradiation, the inclusion of gastric juice from normal rats having no effect on the rate. A severe interference in the absorption and retention of p.o. ^{57}Co-B_{12} was also observed in the irradiated rats: this was evidenced by low serum B_{12} levels, decreased organ uptake of B_{12}, and increased B_{12} excretion. However, when the vitamin was administered i.p., its uptake by various organs was not affected by x-irradiation. Atrophy of the intestinal cells in the animals maintained on a low-protein diet accentuated the adverse effects of radiation on B_{12} absorption; vitamin A deficiency had no appreciable effect on B_{12} absorption by the x-irradiated animals. A sharp drop in viable intestinal flora was observed within 24 hr after irradiation, but their numbers

were found to increase after 3 days. The results suggest that the observed morphological degeneration of mucosal cells in the x-irradiated animals was the primary reason for the B_{12} malabsorption seen after x-ray exposure.

5599 INTESTINAL BLOOD FLOW: AN EVALUATION BY CLEARANCE OF XENON Xe 133 FROM THE CANINE JEJUNUM. *(E.)* Wilson, S. E.; Hiatt, J.; Winston, M.; Passaro, E., Jr. (VA Wadsworth Hosp., Los Angeles, Calif.). *Arch. Surg.* 110(7):797-801, 1975.

Blood flow to the small bowel was assessed by measuring the clearance of xenon from the canine jejunum. ^{133}Xe was injected into the submucosa and muscularis of the jejunum in five anesthetized male mongrels. A thyroid-uptake type scintillation probe was used to register isotope clearance, the rate of disappearance or "washout curve" being used to determine the tissue blood flow following varying degrees of partial aortic occlusion. The shapes of the plotted washout curves from submucosa and muscularis were characteristic and reproducible, a steep or rapid initial component being consistently followed by a slower component. The calculated xenon clearance rates for both tissues (expressed as half-times for isotope disappearance and plotted as a function of decreasing superior mesenteric artery flow) were characteristically rapid for a broad range of superior mesenteric artery flows (90-600 ml/min). With reduction of the superior mesenteric artery flow beyond 80 ml/min, the tissue clearance of xenon was markedly prolonged. Adequate perfusion of the vascular compartments of the small bowel as measured by xenon clearance was maintained until reduction of the superior mesenteric artery flow beyond 80 ml/min. The data suggest that mesenteric vascular obstruction may be considerable before tissue perfusion is markedly reduced, provided that colonic vascular auto-regulation is similar to that of the jejunum. The clearance of a xenon injection from the intestinal parenchyma may, therefore, represent a useful and readily available tool for use in the diagnosis of vascular insufficiency of the bowel.

5600 IMMUNOGENICITY OF LIPID-CONJUGATED PROTEIN IN THE INTESTINE. *(E.)* Heatley, R. V.; Stark, J. M. (Welsh Natl. Sch. Med., Cardiff). *Immunology* 29(1):143-149, 1975.

Lipid-conjugated human serum albumin (HSA) was tested for its ability to induce antibody production, molecular aggregation, and a tendency to attach to cells in female Wistar rats; the amounts of undigested antigen draining in the intestinal lymph were also measured, as was the anticomplementary activity of the antigens. HSA was acylated with lauroyl chloride; different degrees of fatty acid attachment were achieved by differing the molar ratios of lauroyl chloride to protein. Rats were immunized with untreated or lauroylated HSA by i.m. injection (1 mg followed 28 days later by 100 µg) or by injection into the lumen of the small bowel (10 mg followed 28 days later by 1 mg). *In vitro*, rat peritoneal cell suspensions were incubated for 1 hr with acylated protein or HSA exposed to the solvent mixtures required for acylation but without lauroyl chloride (CHSA). The antibody response to lauroylated HSA was significantly greater than that to untreated HSA or CHSA, the response tending to decrease with increasing degrees of acylation. Similar results were obtained with i.m. injection and injection into the small bowel, although the differences in response obtained with the acylated and nonacylated proteins were less clearly defined in the latter case. The increased immunogenicity of the acylated protein was associated with increased molecular aggregation and cellular adherence, the degree of polymerization and percentage attachment increasing with increased acylation. Acylation also reduced the drainage of undigested antigen through the lymphatics. All acylated proteins showed some anticomplementary activity, which increased with the degree of acylation. The data suggest that if lipid-associated protein with an insufficiently hydrophilic surface is absorbed from the gut, an immunogenic event may ensue which in humans might play a role in the pathogenesis of a variety of inflammatory disorders of the gastrointestinal tract for which an immunological basis has been postulated.

5601 ANTIBACTERIAL MECHANISMS IN THE INTESTINE: ELIMINATION OF V. cholerae FROM THE GASTROINTESTINAL TRACT OF ADULT MICE. *(E.)* Knop, J.; Rowley, D. (Dep. Microbiol., Univ. Adelaide, Australia). *Aust. J. Exp. Biol. Med. Sci.* 53(2):137-146, 1975.

5602 ANTIBACTERIAL MECHANISMS IN THE INTESTINE: ELIMINATION OF V. cholerae FROM THE INTESTINES OF INFANT MICE AND THE ROLE OF ANTIBODY. *(E.)* Knop, J.; Rowley, D. (Dep. Microbiol., Univ. Adelaide, Australia). *Aust. J. Exp. Biol. Med. Sci.* 53(2):147-154, 1975.

5603 GASTROINTESTINAL HORMONES AND NEURAL INTERACTION WITHIN THE CENTRAL NERVOUS SYSTEM. *(E.)* Dafny, N.; Jacob, R. H.; Jacobson, E. D. (Univ. Texas Med. Sch., Houston). *Experientia* 31(6):658-660, 1975.

5604 THE INFLUENCE OF ANTIGENIC STIMULATION ON THE ONTOGENY OF LYMPHOID AGGREGATES AND IMMUNOGLOBULIN-CONTAINING CELLS IN MOUSE BRONCHIAL AND INTESTINAL MUCOSA. *(E.)* Milne, R. W.; Bienenstock, J.; Perey, D. Y. E. (McMaster Univ. Med. Cent., Hamilton, Canada). *J. Reticuloendothel. Soc.* 17(6):361-369, 1975.

5605 HYPEREMIA AS A MEASURE OF VIABILITY IN REVASCULARIZED ISCHEMIC INTESTINE. *(E.)* Zarins, C. K.; Skinner, D. B.; Rhodes, B. A.; James, Jr., A. E. (Dep. Surg., Univ. Michigan, Ann Arbor). *Surg. Forum* 24:416-417, 1973.

5606 VARIATIONS OF THE ARTERIAL SUPPLY OF THE DUODENUM AND THE PANCREAS IN THE DOMESTIC CAT (Felis domestica BRISSON). *(E.)* Gomercic, H.; Babic, K. (Vet. Fac., Univ. Zagreb, Yugoslavia). *Acta Anat.* 91(2):213-221, 1975.

5607 HOMOGRAFT OF THE SMALL INTESTINE. METABOLIC AND FUNCTIONAL CHANGES CAUSING IRREVERSIBLE SHOCK. *(E.)* Garrido, H.; De Diego, J. A.; Cano, C.; Lucea, C.; Escribano, P. M.; Manzanos, A. (Hosp. Clin. San Carlos, Madrid, Spain). *Int. Surg.* 60(6/7):351-354, 1975.

5608 VASCULARIZATION OF THE RECTO-ANAL SEGMENT. *(It.)* Chiampo, L.; Percudani, M.; Scarpelli, F.; Paroni, R. (Inst. Pathol., Univ. Parma, Italy). *Ateneo Parmense* [*Acta Biomed.*] 45(3):149-169, 1974.

5609 ACTIVITY OF CERTAIN ENZYMES OF THE ALIMENTARY CANAL AND OF THEIR INHIBITORS UNDER THE ACTION OF RADIATION AND THIOPHOSPHAMIDE. *(Rus.)* Savitsky, I. V.; Korpovich, G. A. (N.I. Pirogov Med. Inst., Odessa, USSR). *Radiobiologiia* 15(1): 120-124, 1975.

5610 STUDIES ON PROTEINASE LEVEL IN GASTROINTESTINAL TRACT AND DIGESTIVE GLANDS IN PYRIDOXINE DEFICIENT ANIMALS. *(E.)* Ghose, C. R.; Ghosh, A. K. (Dep. Biochem., Univ. Calcutta, India). *Jpn. J. Exp. Med.* 45(2):101-104, 1975.

5611 THE EFFECTS OF TEMPERATURE AND 2,4-DINITROPHENOL ON TRANSMEMBRANE POTENTIALS OF SUBMANDIBULAR ACINAR CELLS. *(E.)* Creed, K. E.; McDonald, I. R. (Dep. Physiol., Monash Univ., Clayton, Victoria, Australia). *J. Physiol.* 247(3):521-535, 1975.

5612 THE INFLUENCES OF CHANGES IN TEMPERATURE ON INTESTINAL LYMPH FLOW AND RELATIONSHIP TO OPERATIONS FOR CARCINOMA OF THE INTESTINE. *(E.)* Ackerman, N. B. (Upstate Med. Cent., Syracuse, N.Y.). *Surg. Gynecol. Obstet.* 140(6):885-888, 1975.

5613 CO-CARCINOGENESIS IN THE RAT GASTROINTESTINAL SYSTEM. *(E.)* Vogel, Jr., H.H.; Sebes. J. I. (Univ. Tennessee Cent. Health Sci., Memphis). *Radiat. Res.* 62(3):596-597, 1975.

5614 OPSONIC CHARACTERISTICS OF CHYLOMICRON-BOUND PROTEIN. *(E.)* Mayron, L. W.; Tonaki, H.; Kaplan, E. (VA Hosp., Hines, Ill.). *Proc. Soc. Exp. Biol. Med.* 149(1):248-253, 1975.

See also, 5217, 5219.

DIAGNOSTIC PROCEDURES

5615 ELECTROCOAGULATION BIOPSY WITH SPECIAL REFERENCE TO HISTOLOGIC DIAGNOSIS OF GASTRIC LIPOMA. *(E.)* Yoshida, T.; Nakamura, Y.; Nakamura, K.; Watanabe, H.; Tanaka, K. (Med. Coll. Miyazaki, Japan). *Gastrointest. Endosc.* 21(4):156-158, 1975.

The applicability of electrocoagulation biopsy to submucosal tumors of the stomach was illustrated in the histological diagnosis of two gastric lipomas. The two cases were initially diagnosed as submucosal tumor by roentgenologic and endoscopic examinations, with a negative result by routine biopsy, but were later proved to be lipoma by means of electrocoagulation biopsy. Submucosal tissue from intramural tumors could be obtained by biopsy through the ulcerative lesion several days after electrocoagulation. Electrocoagulation biopsy is recommended for making a histologic documentation of tumors covered by intact mucosa or of small tumors found incidentally; it is considered simple, safe, and easy.

5616 DEVELOPMENT OF AN INTRAVENOUS BILE ACID TOLERANCE TEST: PLASMA DISAPPEARANCE OF CHOLYLGLYCINE IN HEALTH. *(E.)* Korman, M. G.; LaRusso, N. F.; Hoffman, N. E.; Hofmann, A. F. (Mayo Clin., Rochester, Minn.). *N. Engl. J. Med.* 292(23): 1205-1209, 1975

The plasma disappearance of i.v. injected cholylglycine was studied in man by a radioimmunoassay to develop an i.v. bile acid tolerance test. Cholylglycine (1-7.5 mg/kg) was injected into six healthy men at 3 hr intervals at three different dosages to determine the optimal dose; 5 μM/kg was selected for subsequent tests. Forty-five healthy subjects were then injected with the optimal dose of cholylglycine and serial samples of venous blood were collected for radioimmunoassay, specific for conjugates of cholic acid. Cholylglycine disappearance was rapid (2.6 min half-life); the rate of disappearance was independent of dose between 2 and 15 μM/kg. The conjugates of cholic acid increased 10-fold, but the concentration of those bile acids returned to 1 μM by 10 min. No side effects were observed. The results suggest that cholylglycine may be an ideal substance for an i.v. tolerance test for defining hepatic functional mass and that its plasma disappearance may provide the basis for a convenient, safe and sensitive test for detection of liver disease.

5617 CASCADE STOMACH: ROENTGEN APPEARANCE AND SIGNIFICANCE. *(E.)* Keller, R. J.; Khilnani, M. T.; Wolf, B. S. (Mt. Sinai Hosp., New York, N.Y.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(4):746-754, 1975.

Cascade stomach was precisely described so that it can be recognized in the erect position at the onset of a barium series. In the erect position, the presence of a posterior "ridge" which delineates a boundary between the fundus and body of the stomach was used to define the presence of a cascade configuration. Such configuration of the stomach, which is seen in the absence of significant pathologic abnormality, is most often associated with a gas distended distal transverse colon or proximal limb of the splenic flexure. True physiological or normal cascade is not very common; patients with a cascade stomach have demonstrated a variety of lesions, both intrinsic and extrinsic, primarily related to the posterior wall of the stomach. It is suggested that a cascade stomach should not be considered physiologic or within normal limits until a detailed examination of the area is performed.

5618 ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY IN PATIENTS WITH GASTRECTOMY AND GASTROJEJUNOSTOMY (BILLROTH II). A CASE FOR THE FORWARD LOOK. *(E.)* Katon, R. M.; Bilbao, M. K.; Parent, J. A.; Smith, F. W. (Univ. Oregon Med. Sch., Portland). *Gastrointest. Endosc.* 21(4):164-165, 1975.

The use of a fiberscopic endoscope with a forward-viewing optical system was described for cannulation through the afferent loop of a Billroth II anastomosis. Twelve procedures were performed on 10 patients with a distal gastrectomy and Billroth II gastrojejunostomy. A side-viewing fiberduodenscope was used in four patients. Delays and difficulties in identifying and entering the afferent loop and in introducing the instrument through the loop to the papilla of Vater were encountered with all four patients. With the forward-viewing fiberscope used in six patients, the afferent loop was easily identified, entered, and followed to its blind termination. The authors recommend use of the forward-viewing instrument for endoscopic cannulation of the duodenal papilla in patients with gastrojejunostomy but urge particular caution in endoscopy of the afferent limb.

5619 ENDOSCOPIC DIAGNOSIS OF GASTRIC CANCER WITH DYE SCATTERING. *(E.)* Ida, K.; Hashimoto, Y.; Takeda, S.; Murakami, K.; Kawai, K. (Murakami Mem. Hosp., Kyoto, Japan). *Am. J. Gastroenterol.* 63(4):316-320, 1975.

A dye scattering technique was used for the diagnosis of early cancer of the stomach. The examination was performed on 41 patients with early and advanced gastric cancer; indigocarmine was used in 33 cases, methylene blue in two patients, and both dyes in four cases. With routine endoscopy, 35/41 cancerous lesions were diagnosed correctly; with the dye technique, 40/41 lesions were correctly diagnosed. The morphology of the gastric mucosal surface was well defined by the dye scattering technique. In 12 lesions, the whole contour of the cancerous lesion was detected by both methods; in 20/29 lesions, the borders could be completely outlined with dye scattering but only partially defined by routine endoscopy. The dye technique allowed for delineation of minimal depressions in the gastric mucosa; depressions were seen in 12/15 patients in whom no lesion was recognized by routine endoscopy. A correlation was observed between degree of abnormality of surface structure and the depth of cancer infiltration. The indirect dye scattering method

is considered helpful in the clinical diagnosis of gastric cancer by intensifying and delineating mucosal surface alterations.

5620 VALIDITY AND SENSITIVITY OF AN INTRAVENOUS BILE ACID TOLERANCE TEST IN PATIENTS WITH LIVER DISEASE. *(E.)* LaRusso, N. F.; Hoffman, N. E.; Hofmann, A. F.; Korman, M. G. (Mayo Clin., Rochester, Minn.). *N. Engl. J. Med.* 292(23):1209-1214, 1975.

A bile acid tolerance test, which measures the plasma disappearance rate of injected cholylglycine by radioimmunoassay, was studied in 36 patients with biopsy-proved chronic liver disease and compared to other conventional liver function tests. The patients were divided into two groups: one group of 25 patients had abnormal results on conventional liver function tests and were used to validate the sensitivity and reliability of the test; the other group of 11 patients had liver test results within normal limits and were used to compare the sensitivity of the cholylglycine tolerance test with that of other tests and of the fasting-state conjugated cholic acid test. Cholylglycine (5 μM/kg, i.v.) was injected into each patient and serial samples of blood were collected for determinations of conjugated cholic acid values by radioimmunoassay. In the first group of 25, fasting-state levels of conjugated cholic acid were increased in 20, but plasma disappearance of cholylglycine was delayed in all. In the other group, fasting-state levels of conjugated cholic acid were increased in 3/10, but cholylglycine disappearance was delayed in 9/11. The results suggest that the bile acid tolerance test indicates liver disease more sensitively than the fasting-state level of conjugated cholic acid, which, in turn, is more sensitive than other conventional liver tests.

5621 ^{131}I ROSE BENGAL LIVER SCANNING IN THE DIFFERENTIAL DIAGNOSIS OF NEONATAL JAUNDICE. *(E.)* Wicksman, R. S.; Adcock, D. F.; Bream, C. A.; Scatliff, J. H. (Univ. North Carolina Sch. Med., Chapel Hill). *South. Med. J.* 68(5):599-602, 1975.

The radioiodinated rose bengal abdominal scan was used as the only radioactive testing procedure in diagnosis of congenital biliary atresia in infants, and a count ratio was obtained from the scan data as an additional physiologic measurement. The 21 infants were premedicated with stable iodine, then given 10-30 μCi ^{131}I rose bengal i.v. in rough proportion to body weight. Sequential, anterior abdominal scans were usually obtained at 15 min, 1, 3, 6, 24, and 48 hr after injection. Some lateral scans were also done. Count ratio was determined by marking the point of maximal activity, then recording the 24-hr count over this point and dividing it by the maximal count rate. This indicated degree of retention of the radioisotope in the liver, and varied directly with severity of obstruction. Of 22 scans done on 21 infants, diagnosis based on scan only was incorrect in four instances (82% accuracy). In three of these, severity of the obstruction was overestimated. Eight infants were found to have biliary atresia, and 13 intrinsic liver disease, by open or needle biopsy, autopsy, or clinical course. Those with total obstruction had a mean count ratio of 0.76, while those with intrinsic liver disease had one of 0.45. It is concluded that the rose bengal scan alone had an accuracy of 82%, and that determination of count ratio, at no additional expense, would have improved the accuracy.

5622 FIBERCOLONSCOPY FOR THE DIAGNOSIS OF GRANULOMATOUS COLITIS. *(E.)* Watanabe, H.; Narasaka, T.; Imai, H.; Uezu, J. M.; Numazawa, M.; Shoji, K.; Ishikawa, M.; Yamagata, S. (Tohoku Univ. Sch. Med., Sendai, Japan). *Tohoku J. Exp. Med.* 115(3):285-296, 1975.

The pathological, radiological, endoscopic, and clinical differences between granulomatous colitis and ulcerative colitis were studied to test the effectiveness of fibercolonoscopy in diagnosis of granulomatous colitis. Observations were made on 12 cases of granulomatous colitis and 23 cases of ulcerative colitis. Granulomatous colitis showed cobblestone-like or gyrous lesions, segmentally distributed along the bowel, excepting the rectum. The lesions lacked hemorrhage or suppuration, showed microscopic evidence of transmural inflammation and non-caseating granuloma formation, and showed fissuring and stricture. Characteristic were negative tuberculin tests, diarrhea with no bleeding, palpable abdominal mass, and small bowel involvement In contrast, ulcerative colitis showed continuous, symmetrically distributed lesions, which included the rectum. Lesions were rough or granular, or showed pseudopolyposis, hemorrhage, and suppuration. There was no evidence of non-caseating granuloma formation, fissuring, fistula, or stricture. Blood in the stool and lack of small bowel involvement were characteristic. Fibercolonoscopy combined with biopsy is the only procedure that allows direct observation of the lesions. It is concluded that fibercolonoscopy is quite valuable in diagnosis of granulomatous colitis.

5623 THE "RECTO-SIGMOID INDEX". A MEASUREMENT FOR THE EARLY DIAGNOSIS OF HIRSCHSPRUNG'S DISEASE. *(E.)* Pochaczevsky, R.; Leonidas, J. C. (Mt. Sinai Hosp. Serv., City Univ. New York, N.Y.). *Am. J. Roentgenol. Radium Ther. Nucl. Ther.* 123(4): 770-777, 1975.

Barium contrast studies done on 21 newborns with biopsy-proven Hirschsprung's disease, 10 with the meconium plug syndrome, 12 normal term newborns and five with other gastrointestinal diseases were reviewed. The ratio of the widest diameter of the rectum at a level below the third sacral vertebra to that of the sigmoid loop ("recto-sigmoid index") ranged from 0.7 to 0.9 (mean=0.8) in 10 Hirschsprung's disease patients who had no definite colonic transition segment. In contrast, the indices of normal subjects ranged from 1.0 to 1.9 (mean=1.4) and those of newborns with other disease of the gastrointestinal tract ranged from 1.3 to 1.5 (mean=1.4).

Based on these results, it is suggested that the "recto-sigmoid index" may aid in the diagnosis of Hirschsprung's disease in the newborn without a definite transition segment on the barium contrast study.

5624 DETERMINATION OF SERUM LIPOPROTEIN-X FOR THE EARLY DIFFERENTIATION BETWEEN NEONATAL HEPATITIS AND BILIARY ATRESIA. *(E.)* Campbell, D. P.; Poley, J. R.; Alaupovic, P. (Univ. Oklahoma Health Sci. Cent., Oklahoma City). *J. Surg. Res.* 18(4):385-390, 1975.

A combination of lipoprotein-X determination and cholestyramine treatment was used to differentiate between neonatal hepatitis and biliary atresia. Infants (28) with cholestatic jaundice of etiology not determinable by standard testing had serum lipoprotein-X measured after a 6-hr fast. Those with a positive reaction were given 1 g cholestyramine/day p.o. for 2 weeks, followed by a second lipoprotein-X determination. Of the 28 infants, 14 proved to have biliary atresia on surgical exploration. All 14 were positive for lipoprotein-X. Of these, 13 showed a rise in serum lipoprotein-X levels after the cholestyramine treatment. The serum from the 14th was in transit for 4 days, sufficient time for the lipoprotein-X to dissociate. The remaining 14 infants had intrinsic cholestasis, 12 from neonatal hepatitis. Of the 14, five were positive for lipoprotein-X. After cholestyramine treatment, the lipoprotein-X levels dropped significantly. Six of the nine negative for lipoprotein-X were given the cholestyramine treatment; all remained negative. Of the 28 infants, 27 were diagnosed accurately (96.4%). It is concluded that lipoprotein-X determination coupled with cholestyramine treatment is an accurate and simple method of distinguishing biliary atresia and intrinsic cholestasis.

5625 EMERGENCY PERITONEOSCOPY. *(Ger.)* Baerlocher, C.; Engelhart, G.; Fahrlander, H. (Dep. Med., Univ. Basel, Switzerland). *Leber Magen Darm* 3(1):11-14, 1973.

Emergency peritoneoscopy was introduced in the authors' clinic in 1964 and to date 180 such examinations were performed corresponding with 10% of the total number of laparoscopies. Endoscopic results, shown in tabular form, covered 100 patients with blunt abdominal trauma, including 47 patients with free blood in the abdominal cavity and one with urine due to rupture of the bladder; further, 80 patients without abdominal trauma involving peritonitis, mesenterial infarct, pancreatitis and miscellaneous causes. Data are given on confirmation of the endoscopic findings with clinical course, surgery or autopsy. While the method, described as far back as 1937 and 1942, has not found general acceptance, the authors consider emergency peritoneoscopy a safe procedure, quick to carry out (normally in 10-15 min), tolerated even by seriously ill patients, and of high diagnostic value. In some aspects it may be preferable to other diagnostic procedures such as exploratory puncture or peritoneal lavage. An experienced endoscopy team, however, is a prerequisite.

5626 THE ROLE OF GLUCAGON IN ENDOSCOPIC CHOLANGIOPANCREATOGRAPHY. *(E.)* Silvis, S. E.; Vennes, J. A. (Minneapolis VA Hosp., Minn.). *Gastrointest. Endosc.* 21(4):162-163, 1975.

The effectiveness of glucagon in control of duodenal motility, in promoting efficiency in performing endoscopic cholangiopancreatography, and in preventing pancreatitis was determined in a double-blind study of 55 patients. All patients received dicyclomine (60 mg, i.m.) and atropine (0.4 mg, i.m.) before the procedure. When the duodenoscope was in position, 28 patients received 1 ml glucagon and 27 patients received 1 ml saline. Change in motility was evaluated and urine amylase was obtained. A marked subjective change in motility was noted in 8/17 patients who had received glucagon compared with 2/16 in the placebo group. Twenty-four hours after the procedure, 8% of the patients who had received glucagon and 64% of the placebo patients had an elevated urine amylase value; the mean urinary amylase value was 186 U/hr in glucagon patients and 682 U/hr in placebo patients (statistically significant). One patient who had received glucagon developed cholangitis, and one placebo patient developed mild pancreatitis. The results suggest that glucagon is a safe and valuable means of controlling duodenal motility in endoscopic pancreatocholangiography.

5627 NEW DIAGNOSTIC AND THERAPEUTIC TECHNIQUES IN THE MANAGEMENT OF PYOGENIC LIVER ABSCESSES. *(E.)* Ranson, J. H. C.; Madayag, M. A.; Localio, S. A.; Spencer, F. C. (New York Univ. Med. Cent., New York, N.Y.). *Ann. Surg.* 181(5):508-518, 1975.

The treatment of 14 patients (12 men and 2 women, aged 22-80 yr) operated on for pyogenic liver abscesses was studied. Eight cases (57%) were cryptogenic. Other abscesses were associated with biliary disease (3), abdominal sepsis (2), and trauma (1). Abscesses were present on hospitalization in 12 patients. Clinical findings included fever (101-108 F), 100%; leucocytosis, 71%; anorexia and vomiting, 50%; localized tenderness and hepatomegaly, 50%; hypoalbuminemia, 86%; hypocholesterolemia, 78%; elevated SGOT, 71%; and elevated alkaline phosphatase, 43%. Technetium hepatic scintiscans showed focal defects in 10 of 12 patients (83%), but did not detect multiple abscesses in 2 of these. Hepatic arteriography performed in 10 patients was highly accurate, outlining single abscesses in 6 and multiple abscesses in 4. Furthermore, in one patient a false positive scintiscan was demonstrated by negative arteriography, confirmed by autopsy. In 4 patients, arteriography indicated an abscess in the posterior-superior area of the right hepatic lobe. With precise anatomical localization, a transthoracic approach permitted uncomplicated drainage in each case. This approach provided excellent exposure and direct drainage for abscesses in this area. An additional therapeutic adjunct in 2 patients, with 4 and 11 abscesses each, was postoperative intraportal infusion of antibiotics through the umbilical vein. Thirteen patients (83%) recovered, one dying from pulmonary embolism. Primary

hepatic abscesses occur with increasing frequency. The methods described allow more precise preoperative diagnosis and direct surgical drainage.

5628 SUPRAGASTRIC PANCREOSCOPY: LAPAROSCOPY AND BIOPSY OF THE BODY OF THE PANCREAS. *(Ger.)* Meyer-Burg, J.; (Steglitz Clin., Free Univ., Berlin, Germany). *Fortschr. Med.* 91(26):1017-1019, 1975.

A report on the direct laparoscopic inspection of the pancreas and its possible value as a diagnostic tool in pancreatic diseases was presented. The operative procedure in performing a laparoscopy was briefly described and visual characteristics of the pancreas were noted in the normal state, in acute and in chronic pancreatitis, and with neoplasms of the pancreas, which are often associated with liver metastases, Courvoisier's law or cholestasis. Clinical data, cited from other publications, included 125 laparoscopies which allowed inspection of parts of the pancreas in 81 (65%) cases. These included 15 patients with pancreatic neoplasms, six patients with chronic pancreatitis, one patient after passage of acute pancreatitis, and one patient with a pancreatic pseudocyst falsely diagnosed as a tumor. In 31 cases material was removed for cytological examination, usually by multiple puncture of each organ in different locations. Admittedly, the limitations of the method reside in the fact that only protions of the pancreas (largely the surface) are accessible to inspection. Also, it does not usually serve in early recognition of a pancreatic neoplasm since most patients do not seek medical help in the beginning stage of the disease. Its chief value, therefore, is its application within the general framework of diagnosing pancreatic diseases.

5629 RADIOSELENUM PANCREOZYMIN-SECRETIN TEST AS A CLINICAL TEST FOR PANCREATIC EXOCRINE FUNCTION. *(E.)* Shichiri, M.; Etani, N.; Yoshida, M.; Harano, Y.; Hoshi, M.; Shigeta, Y.; Abe, H. (Osaka Univ. Med. Sch., Japan). *Am. J. Dig. Dis.* 20(5):460-468, 1975.

The appearance of radioselenium in the protein fraction of duodenal aspirates following stimulation with pancreozymin and secretin was investigated. Pancreatic exocrine function was determined in 36 subjects: 17 were classified as normal and 19 had pancreatic diseases including chronic pancreatitis (14), pancreatic carcinoma (3), and pancreatic cyst (2). A good distinction between normal subjects and patients with pancreatic disease was obtained by measuring ^{75}Se-radioactivity in the protein fraction of duodenal aspirates; either cumulative radioactivity during the combined 80 min post-pancreozymin-secretin period or maximum ^{75}Se-specific activity during the postsecretin period was used as an index. It is suggested that this test may be useful and sufficiently reliable for detecting abnormal pancreatic exocrine function. This test can be performed simultaneously with the conventional pancreozymin-secretin test, provocative serum enzyme response to pancreozymin and secretin stimulation, and pancreatic scintiscanning. Inclusion of the cytology test may help to distinguish carcinoma from other pancreatic diseases. The combined test may provide more accurate information on pancreatic exocrine function.

5630 CRITICAL EVALUATION OF HEPATIC SCINTIANGIOGRAPHY FOR NEOPLASTIC TUMORS OF THE LIVER. *(E.)* Stadalnik, R. C.; DeNardo, S. J.; DeNardo, G. L.; Raventos, A. (Univ. California Sch. Med., Davis, Sacramento Med. Cent.). *J. Nucl. Med.* 16(7):595-601, 1975.

Dynamic hepatic scintiangiography was compared with histologic and radiopaque diagnostic techniques in the evaluation of suspected hepatic neoplasms in humans. In 92 patients previously diagnosed as having hepatic metastases or normal livers, histologic examination of the liver was performed within 2 months of hepatic scintigraphy and scintiangiography; in 10 of these patients, radiopaque arteriography was also performed. Of the 92 patients studied, 23 had histologically normal livers, 10 had hepatomas, and 59 were classified as having hepatic metastases. In all 10 patients with hepatoma, the scintiangiographic and histologic observations agreed; 9 of these patients had a "tumor stain." Scintiangiograms in 51 of the patients with metastases also showed a tumor stain, and in 2 of the metastasis patients, scintiangiography revealed tumor vascularity where the scintigraphic results were normal. In 2 of 4 patients with metastases and 2 of 6 patients with hepatomas, scintiangiograms revealed tumor stain which was not evident on radiopaque arteriography. In 20 of the patients with normal hepatic tissue, both the hepatic scintigram and corresponding scintiangiogram were normal, while in 2 others both were abnormal; in 1 other patient, the scintigram was normal, but the scintiangiogram demonstrated focal arterialization due to a renal carcinoma. The results indicate that: a normal scintigram with a corresponding normal scintiangiogram make neoplastic involvement of the liver improbable; a normal scintigram and a tumor stain on the scintiangiogram of a patient with a known primary neoplasm outside the liver is suggestive of hepatic metastases; and neoplastic arterialization or tumor stain is more readily detected by scintiangiography than by radiopaque arteriography. Dynamic hepatic scintiangiography is a simple technique which should be included in the evaluation of the liver for neoplastic involvement.

5631 ABRASIVE CYTOLOGY OF THE PANCREATIC AND BILIARY DUCT IN MAN. *(E.)* Weidenhiller, S.; Fluegel, H.; Rosch, W. (Dep. Med., Univ. Erlangen-Nuremberg, Germany). *Endoscopy* 7(2):72-74, 1975.

Abrasive cytological examination of the pancreatic and biliary duct was evaluated as a technique for the preoperative diagnosis of malignant tumors of the pancreas and biliary tree. A specially designed Fogarthy-catheter was run through a conventional endoscopic retrograde cholangiopancreatography tube

and introduced into Vater's papilla in 25 patients; after positioning, the abrasion balloon at the tip of the catheter was inflated and withdrawn. Using this probe, abraded cell material was obtained from the pancreatic duct in 19 patients and from the common bile duct in 6 patients. Sufficient cytological material was obtained in almost all patients without noticeable complications. In 1 patient in whom a previous retrograde pancreatogram was suspicious of pancreatic carcinoma, the diagnosis was cytologically confirmed prior to surgery. The abrasion balloon technique described will probably be of value in the cytological examination of the common bile and pancreatic ducts, and will be particularly useful in cases of suspicious strictures or complete obstruction of the ductal system by malignancy.

5632 DIAGNOSTIC VALUE OF SERUM BILE ACID ESTIMATIONS IN LIVER DISEASE. *(E.)* Barnes, S.; Gallo, G. A.; Trash, D. B.; Morris, J. S. (R. Free Hosp., London, England). *J. Clin. Pathol.* 28(6):506-509, 1975.

Fasting and postprandial serum bile acid concentrations were measured in normal (10 men and 8 women, aged 23-35 yr) subjects and in patients (18 men and 12 women, aged 25-65 yr) with a variety of hepatobiliary diseases. The 30 patients were histologically diagnosed as follows: cryptogenic cirrhosis (9), alcoholic cirrhosis (3), primary biliary cirrhosis (6), active chronic hepatitis (4), cholestatic syndromes (drug jaundice) (3), viral hepatitis (2), and large bile duct obstruction (3). In addition, 7 persons (5 men and 2 women, aged 18-75 yr) with hepatovenous outflow block (3) and unconjugated hyperbilirubinemia (4) were studied. In normal subjects the serum bile acid concentration ranged between 0 and 15 μM/liter. No differences were noted between the sexes or between fasting and postprandial concentrations. In 27 of the 30 patients with hepatobiliary disease, fasting concentrations were above the normal range (22-600 μM/liter). In 3 subjects with liver disease the fasting serum bile acid concentrations were normal, but 2 hr after the fatty breakfast they became markedly elevated. In 2 of these 3 subjects, other biochemical estimations (serum bilirubin, aspartate transaminase, alkaline phosphatase) were normal. Compared with the other biochemical estimations the postprandial serum bile acid concentrations were the only estimations which were consistently abnormal. In the 4 patients with unconjugated hyperbilirubinemia, both fasting and 2 hr postprandial bile acid concentrations were normal as were the liver function tests, which was expected as they had no histological liver disease. Two of the 3 with hepatic venous outflow block had normal fasting concentrations, but all had elevated postprandial serum bile acid concentrations, indicating a degree of hepatocellular dysfunction. This study shows that the 2-hr postprandial serum bile acid concentrations are elevated in patients with histological hepatobiliary disease, even when fasting concentrations and other liver function tests are normal. Despite the obvious value of serum bile acids in indicating the presence of histological liver damage, the results indicate that such measurements are of no value in distinguishing between particular liver diseases, particularly in deciding whether cholestasis is of intra- or extrahepatic origin.

5633 THE USE OF A SHORT-LIVED RADIONUCLIDE IN THE INVESTIGATION OF ACUTE BLEEDING IN THE UPPER GASTROINTESTINAL TRACT. *(E.)* Pettersson, C.; Forssman, O.; Hollender, A.; Mulder, J. L. (Cent. Hosp., Boras and Ferring AB, Malmo, Sweden). *Scand. J. Gastroenterol.* 10:213-215, 1975.

The possibility of detecting and monitoring bleeding in the upper gastrointestinal tract using a short-lived radionuclide $^{113}In^{m}$ was investigated. *In vitro* studies were performed to confirm the protein binding of indium ions. *In vivo* studies in 2 healthy subjects showed that when the radionuclide was ingested in 0.04 M HCl, no absorption took place. The radionuclide was passed along the gastrointestinal tract and no significant binding to the stomach or the duodenum could be detected. The procedure was then studied in 28 patients with acute bleeding in the upper gastrointestinal tract. In 16 patients no signs of blood could be detected from aspirates from the duodenum or stomach. In another 4 cases, only traces of blood could be found. In the remaining 8 cases, the aspirate showed a +++ Weber reaction. In 11 cases with a positive, and in 1 case with a negative, test for blood in the aspirate, $^{113}In^{m}$ was injected i.v. In no patient was a higher radioactive count found in the aspirates than in controls. In all patients, tests for blood in the feces remained positive for 2 weeks or less. Current bleeding was noted in only 1 patient as judged by the injection of $^{113}In^{m}$. By using this radionuclide to estimate the amount of current bleeding in the gastrointestinal tract, bleeding in the amount of 8-10 ml/hr can be detected, providing a complete collection of the contents of the gastrointestinal lumen can be performed. As the radionuclide is short-lived it can be used repeatedly in the same patient. The most striking finding of this study was that in patients with acute bleeding in the upper gastrointestinal tract but without signs of shock, the bleeding mostly ceases within a very short time after onset. Therefore, the blood in aspirates from the duodenum or stomach is obviously mostly of somewhat older origin. If bleeding is monitored by this present method, it might be possible for a patient to leave the hospital long before a negative blood test in the feces is obtained.

5634 TECHNETIUM-99m-PYRIDOXYLIDENEGLUTAMATE: A NEW HEPATOBILIARY RADIOPHARMACEUTICAL. II. CLINICAL ASPECTS. *(E.)* Ronai, P. M.; Baker, R. J.; Bellen, J. C.; Collins, P. J.; Anderson, P. J.; Lander, H. (Inst. Med. Vet. Sci., R. Adelaide Hosp., South Australia). *J. Nucl. Med.* 16(8):728-737, 1975.

The new radiopharmaceutical, Technetium-99m-pyridoxylideneglutamate (^{99m}Tc-PG), was clinically evaluated in ten control subjects, 24 patients referred for the investigation of right upper quadrant abdominal pain, and 36 patients referred for investigation of jaundice. No patient preparation or premedication was used prior to the i.v. administration of 5 to 6 mCi of the compound. Sequential scintigrams were

recorded for all patients, and contrast radiologic studies of the biliary tree were performed as indicated. There were no subjective symptoms or biochemical changes associated with the administration of the radiopharmaceutical. In the control subjects, the biliary tree and gallbladder were seen within 10 to 15 min of injection and within 20 min marked accumulation of radioactivity was noted in the gallbladder and gastrointestinal tract; the ^{99m}Tc-PG-cholescintigrams were normal in seven controls and the gallbladder was not visualized in the remaining three. Gallbaldder disease was present in 13 of the patients referred for investigation of right upper quadrant pain. All seven patients with acute cholecystitis and one out of four patients with chronic cholecystitis had nonvisualization of the gallbladder, whereas five patients with chronic cholecystitis and/or cholesterolosis had normal cholescintigrams; in the first group, nonvisualization of the gallbladder was also reported on the contrast studies. Cholescintigraphy was greatly inferior to contrast radiologic studies in the detection of gallbladder stones. Diagnosis of complete extrahepatic biliary obstruction was correctly made by the cholescintigram in all 11 cases in which it occurred, whereas correct diagnosis based on the cholescintigram was made in only seven out of 14 patients with incomplete extrahepatic biliary obstruction. A diagnosis of obstruction due to malignancy was correctly made in eight out of ten patients by means of a scintigraphic equivalent to Courvoisier's sign. A nonspecific pattern, consistent with incomplete biliary obstruction or hepatocellular disease, was observed on the cholescintigram in all 11 patients with hepatocellular disease. It is suggested that the ^{99m}Tc-PG-cholescintigram be used in conjunction with contrast radiologic studies in the preoperative investigation of patients with possible surgical disease of the biliary tract; the former is more appropriate in the patient with jaundice while the latter is more appropriate in the nonjaundiced patient.

5635 AN EXPERIMENTAL STUDY OF ^{99m}Tc PERTECHNETATE ABDOMINAL SCANS IN JEJUNAL INTUSSUSCEPTION: PRELIMINARY RESULTS. *(E.)* James, A. E., Jr.; Strecker, E. P.; Miller, F. J., Jr.; Bush, M. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *J. Surg. Res.* 19(1):71-76, 1975.

The abnormalities noted on ^{99m}Tc pertechnetate abdominal scans of dogs with small bowel intussusceptions were studied. Surgical, nonreducible small bowel intussusceptions were produced in eight mongrels. At various intervals thereafter, ^{99m}Tc pertechnetate (75 μCi/kg) was administered i.v. and abdominal scans were obtained 30 min, 4 hr, and 24 hr later using a rectilinear scanner. Scans were also obtained on normal controls receiving ^{99m}Tc pertechnetate for other diagnostic studies. The animals were sacrificed 12, 24, or 48 hr after injection, the small bowel was removed, and radiographs were made. Samples of the intraluminal fluid and tissue samples were counted in a well counter and histological preparations were made. A characteristic abnormal image was seldom obtained on the abdominal scans, and even in animals giving positive results, the bowel proximal to the intussusception, not the intussusception itself, was visualized. In addition, the major concentration of radioactivity was in the intraluminal fluid proximal to the surgically-induced intussusception. Selective concentration of the radiopharmaceutical was also observed just proximal to a variety of surgically-created mechanical obstructions of the jejunum and ileum. The scans on normal dogs showed accumulation of radioactivity in the stomach and urinary bladder as well as occasional visualization in the region of the duodenum. Despite catheterization, the urinary bladder was often visualized. The results suggest that the abdominal scan abnormalities in patients with intussusception are not caused by visualization of the lesion but are due to attendant bowel obstruction.

5636 TOMOECHOGRAPHIC DIAGNOSIS OF HEPATIC POLYCYSTOSES. *(Fr.)* Weill, F.; Aucant, D.; Becker, J. C.; Eisenscher, A.; Kraehenbuhl, J. R. (Univ. Hosp. Cent., Besancon, France). *(Ann. Radiol. (Paris)* 17(7):685-689, 1974.

Tomoechography is considered an excellent tool in the diagnosis of hepatic polycystosis due to the specific character of the ultrasonic image of lesions containing echo-free zones. Six cases of hepatic polycystosis (all women) were detected in over 4000 ultrasonic examinations. In four of the patients with discrete hepatomegaly and no palpable cystic formation, hepatic polycystosis was detected by additional ultrasonic examination following the diagnosis of renal polycystosis. In the two other patients, who exhibited marked hepatomegaly, renal polycystosis was discovered secondarily. The association of renal polycystosis is evidently an important positive clue in the diagnosis of hepatic polycystosis.

5637 EXPERIMENTAL AND CLINICAL STUDIES WITH ^{131}I-BSP. *(Ger.)* Mostbeck, A.; Peschl, L.; Rettenbacher-Taubner, H.; Neumayr, A. (Wilhelmine Hosp., Vienna, Austria). *Z. Gastroenterol.* 12(7):547-556, 1974.

In vitro and *in vivo* studies demonstrated that by using ^{131}I-BSP with a high specific activity, it is possible to reduce the dose of BSP from 5 mg/kg to less than 1 mg and thus to reduce the likelihood of untoward reactions to the dye. *In vitro* studies revealed that ^{131}I-BSP had the same R_f value as inactive BSP on thin-layer chromatography, was chemically stable, and was bound almost quantitatively to serum proteins when they were precipitated with trichloroacetic or sulfosalicylic acids. In *in vivo* studies, there was a good correlation between clearance constants for the first 10 to 12 min and the 45:3-min retention values obtained with both forms of BSP. However, ^{131}I-BSP was excreted somewhat more slowly by the liver. The clearance constant was reduced by about 50% when ^{131}I-BSP and inactive BSP were administered simultaneously. When instilled into the duodenum, only 0.4 to 0.6% of ^{131}I-BSP was later detected in the blood. Although

very small quantities of ^{131}I-BSP were found in the thyroid (maximum of 3-5% of the i.v. administered dose after 24 hr), iodine should be administered to block the thyroid. Good correlations were found between clearance constants and 45:3-min retention values obtained from blood samples and those obtained by measurement of radioactivity over the skull. However, the use of radiation measurements gave consistently smaller clearance constants and significantly higher retention values than were obtained with inactive BSP. These differences are attributed to the distribution of radioactive BSP between vascular and extravascular space. A comparison of clearance constants determined with ^{131}I-BSP on 198 patients (91 with no evidence of liver disease, the rest with fatty liver, acute and chronic hepatitis, compensated and decompensated cirrhosis, obstructive jaundice and intrahepatic cholestasis) gave good separation between normal subjects and those with liver diseases other than fatty liver. Of 30 histologically confirmed cases of fatty liver, only six had pathological values. All patients tolerated i.v. injections of ^{131}I-BSP, and no reactions occurred when the test was repeated after an interval of several days to several weeks.

5638 FUNCTIONAL EXAMINATION OF DEFECATION IN CHILDREN. *(Ger.)* Berger, D.; Landry, M. (Lausanne Univ. Clin., Switzerland). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 121(4):428-434, 1974.

Kinematographic studies of the anorectal canal were performed during defecation on 11 children, aged 6 weeks to 10 yr, using video tape recording and image intensification. In some cases, electromyograms (EMG) were obtained from the puborectal loop and the external anal sphincter to differentiate between myogenic and neurogenic disorders. In constipated children, both under basal conditions and during defecation, anorectal angles were smaller than normal; the anal canal contracted only slightly and was generally conical in shape. The sphincter zone sometimes appeared spastic. In several constipated children, basal activity on the EMG was increased, but no pathognomonic significance was attributed to this finding. In Hirschsprung's disease involving the rectum, changes found in constipated children were more pronounced and were accompanied by dyskinesia in the aganglionic part of the large intestine. In a 10-yr-old boy who developed neurogenic constipation and spastic paresis on the right as the result of a lumbosacral fracture, both the external urethral sphincter and the anorectal region showed spastic changes because both sphincters were innervated by the sacral nerve. Classical anatomical features were totally absent in a 8-yr-old girl with meningomyelocele: the anorectal angle could not be measured, the anal canal was wide open and defecation had to be aided by abdominal pressure. An EMG of the external anal sphincter was almost a straight line. In children with high anorectal malformations, no matter what the clinical findings, the anorectal angle was smaller than in normal children and there was a certain rigidity and inactivity of the anal canal. These radiological measurements gave a rather accurate picture of the quality of continence.

5639 THE PANCREATOGRAPHIC EFFECT DURING PHARMACOANGIOGRAPHY OF THE PANCREAS. *(E.)* Schmarsow, R.; Peters, P. E. (Deutsche Klink Diagnostik, Wiesbaden, Germany). *Acta Radiol. [Diagn.] (Stockh.)* 16(1):73-80, 1975.

The value of pancreatography in distinguishing between chronic pancreatitis and carcinoma of the pancreas was investigated. Secretin (1.5 IU/kg), priscoline (0.7 mg/kg), serotonin (0.7 γ/kg), and caerulein (0.3 γ/kg) were used to enhance the pancreatographic effect. Seventy examinations were performed in 55 adult patients with possible pancreatic disorders. In a series of 174 celiac angiographies performed without drugs, the pancreatographic effect was present in 18.4%. In the 55 angiographies performed with drugs, the effect was produced in 74.5%. By injecting the contrast medium into the hepatic and splenic arteries and in a few cases into the gastroduodenal and dorsal pancreatic arteries without drugs increased the frequency of the pancreatographic effect to 43.3% (out of 30 cases). The use of drugs increased it further to 71.4% (out of 28 cases). With the addition of vasodilatory drugs, higher rates of pancreatographic effect were achieved. In 31 cases the type of pancreatographic effect was compared to pancreatic function. A homogeneous pancreatographic effect was achieved in 14 patients with normal pancreatic function. In six cases with a pathologic test, the pancreas appeared mottled. No correlation between scanning with the isotope ^{75}Se-methionin and the pancreatographic effect was found. The few cases of malignancy in this study material suggest that the pancreatographic effect is completely or partially absent in carcinoma patients. A homogeneous pancreatographic effect seems to exclude a carcinoma and a mottled effect indicates chronic pancreatitis. The absence of pancreatographic effect in carcinoma may be due to the fact that the neoplasm is mostly hypovascular and that the neoplastic tissue has an almost homogeneous dense fibrous stroma, which impedes the accumulation of the contrast medium in this region.

5640 DIAGNOSIS OF STENOSIS OF THE PAPILLA OF VATER. *(Ger.)* Hess, W. (no affil.). *Fortschr. Med.* 93(5):200, 1975.

5641 DIAGNOSIS OF STAGE 0 GASTROINTESTINAL TUMORS: IS THIS A CLINICAL REALITY WITH ENDOSCOPY TECHNIQUES? *(It.)* Di Matteo, S.; Bigotti, A.; Crespi, M. (Queen Elena Inst. Tumor Res. Treatment, Rome, Italy). *Minerva Gastroenterol.* 20(2):57-58, 1974.

5642 ANALYSIS OF THE BSP PLASMA CLEARANCE CURVE WITH COMPARTMENT MODELS. *(It.)* Molino, G.; Milanese, M. (Inst. Med. Semeiotics, Univ. Turin, Italy). *Boll. Soc. Ital. Biol. Sper.* 50(20):1668-1673, 1974.

5643 CHOLANGIOGRAPHY WITH IOTETROXIDE (BC-17) BY CONVENTIONAL INTRAVENOUS INJECTION. *(Jpn.)* Matsuda, T.; Ohnuma, I.; Suga, S.; Isobe, Y.; Shimaji, T.; Koide, A. (Natl. Nagoya Hosp., Japan). *Rinsho Hoshasen* 20(2):127-136, 1975.

5644 COMPRESSION METHOD ON PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY. *(Jpn.)* Nimura, Y.; Hichino, S.; Sato, T.; Kamiya, T.; Ieda, H.; Hayakawa, N.; Nakae, Y.; Kondo, T.; Matsuno, T.; Enomoto, I. (Yachiyo Hosp. Aichi Prefecture, Japan). *Rinsho Hoshasen* 19(12):933-941, 1974.

5645 A STUDY OF ENDOSCOPIC RETROGRADE PANCREATOCHOLANGIOGRAPHY. *(Jpn.)* Urakami, Y.; Kishi, S. (Tokushima Univ. Med. Sch., Japan). *Rinsho Hoshasen* 20(2):111-117, 1975.

5646 ENDOSCOPIC PANCREATGOCHOLANGIOGRAPHY IN THE DIAGNOSIS OF DISEASES OF THE PANCREAS AND BILIARY DUCTS. *(Rus.)* Tofan, A. V.; Demin, M. D.; Temin, V. T.; Shalimov, S. A.; Chernyi, V. A.; Shpontak, A. S. (Dept. Oncol., Kiev Inst. Postgrad. Training Phys., USSR). *Klin. Khir.* (3):12-17, 1975.

5647 SERUM SECRETIN-PANCREOZYMIN TEST IN DIAGNOSING CHRONIC PANCREATITIS. *(Rus.)* Timoshina, I. V. (Hepatopancreat. Dept., All-Union Sci. Res. Inst. Gastroenterol., USSR). *Sov. Med.* (2):73-77, 1975.

5648 THERMOGRAPHY IN ACUTE PANCREATITIS. *(Rus.)* Sukharev, V. F.; Kozlov, O. A. (I. I. Dzhanelidze Leningrad Sci. Res. Inst. First Aid, USSR). *Klin. Khir.* (3):20-24, 1975.

5649 EXAMINATION OF THE ACUTE EFFECT OF PREDNISOLONE ON ACID SECRETION IN THE STIMULATED GASTRIC MUCOSA IN MAN. *(Ger.)* Mertz, D. P.; Papavassiliou, K. (Balneol. Clin., Horn. Bad Meinberg, Germany). *Munch. Med. Wochenschr.* 117(14):575-578, 1975.

5650 RADIOLOGICAL AND ENDOSCOPIC INVESTIGATIONS OF THE ESOPHAGUS IN 33 CASES OF GENERALIZED SCLERODERMA. *(Ger.)* Petite, J. P.; Chaput, J. C.; Fiessinger, J. N.; Housset, E. (Broussais Hosp., Paris, France). *Munch. Med. Wochenschr.* 117(13):525-530, 1975.

5651 THE DIAGNOSIS OF CANCER OF THE STOMACH. *(Fr.)* Vilardell, F. (Santa Cruz y San Pablo Hosp., Barcelona, Spain). *Schweiz. Med. Wochenschr.* 105(18):556-559, 1975.

5652 LACTULOSE: CLINICAL EXPERIENCE, MECHANISM OF ACTION AND ITS USE IN X-RAY DIAGNOSIS OF THE SMALL INTESTINE. *(Cs.)* Pirk, F.; Vosmik, J.; Skalu, I. (Res. Cent. Metab. Nutr., Prague, Czechoslovakia). *Vnitr. Lek.* 20(3):251-257, 1974.

5653 DIAGNOSTIC DIFFICULTIES IN CHOLECYSTITIS AND CHOLANGITIS IN CHILDREN. *(Pol.)* Sowinska, J.; Wierzbolowicz, J.; Rondio, H. (Munic. Child. Hosp., Warsaw, Poland). *Pediatr. Pol.* 50(2):161-167, 1975.

5654 ENDOSCOPY DURING GASTROINTESTINAL SURGERY. *(Fr.)* Doutre, L. P.; Quinton, A.; Gratadour, P. (no affil.). *Bordeaux Med.* 7(16):2329-2332, 1974.

5655 ARTERIOGRAPHY IN THE DIAGNOSIS OF TUMOURS OF THE SMALL INTESTINE. *(Fr.)* Benacerraf, R. (Louis-Mourier Hosp., Colombes, France). *Ann. Radio. (Paris)* 17(8):751-764, 1974.

5656 TUMORS OF THE SMALL INTESTINE: A RADIOLOGICAL PROBLEM. *(Fr.)* Queloz, J.; Realini, S.; Candardjis, G. (Univ. Hosp., Lausanne, Switzerland). *Schweitz. Med. Wochenschr.* 105(18):579-582, 1975.

5657 THE CERULEIN TEST: NEW METHOD FOR EXPLORATION OF EXOCRINE PANCREAS FUNCTION. *(It.)* Zaccherotti, L.; Algeri, R. (Munic. Hosp., Grosseto, Italy). *Minerva Gastroenterol.* 20(2):72-73, 1974.

5658 DOUBLE CONTRAST EXAMINATION OF THE COLON WITH INGESTED BARIUM. *(Jpn.)* Niizuma, S.; Kobayashi, S.; Yamamoto, M. (Cancer Cent. Niigata Prefecture, Japan). *Rinsho Hoshasen* 20(1):57-65, 1975.

5659 DIAGNOSTIC POSSIBILITIES IN DEGENERATIVE GASTRIC POLYPOSIS. *(It.)* Di Silverio, C.; Ottolenghi, G. (Gen. Prov. Hosp., San Remo, Italy). *Minerva Gastroenterol.* 20(2):59, 1974.

5660 THE DIAGNOSIS AND INCIDENCE OF DRUG-INDUCED OR DRUG-ASSOCIATED GASTRIC AND DUODENAL BLEEDING NOT CAUSED BY ESOPHAGEAL VARICES OR HIATUS HERNIA. *(Ger.)* Murr, H.; Feifel, G.; Schramm, W.; Marx, R. (no affil.). *Thromb. Diath. Haemorrh.* (suppl. 61):155-166, 1975.

5661 DIAGNOSIS OF EARLY FORMS OF STOMACH CANCER BY LARGE-FRAME FLUOROGRAPHY. *(Rus.)* Vintergalter, S. F. (N. N. Petrov Res. Inst. Oncol., Leningrad, USSR). *Vestn. Rentgenol. Radiol.* (2):92-94, 1975.

5662 RADIOLOGICAL DIAGNOSIS OF CHOLECYSTOSES. *(Fl.)* Ruijs, J. H. J. (Acad. Hosp., Utrecht, Netherlands). *Ned. Tijdschr. Geneeskd.* 118(46):1754-1759, 1974.

5663 DIAGNOSIS OF THE MALLORY-WEISS SYNDROME. *(Ger.)* Filippini, L.; Nauer, E. (Canton Hosp., Luzerne, Switzerland). *Dtsch. Med. Wochenschr.* 100(24):1341-1343, 1975.

5664 COMPARATIVE STUDY OF SECRETIN AND LUNDH TESTS. *(E.)* Gyr, K.; Agrawal, N. M.; Felsenfeld, O.; Font, R. G. (VA Hosp., New Orleans, La.). *Am. J. Dig. Dis.* 20(6):506-512, 1975.

5665 RESULTS OF PANCREATIC SCINTIGRAPHY WITH SELENOMETHIONE 75: 400 CASES. *(Fr.)* Quinton, A.; Rhahali, A.; Dubarry, J. J.; Blanquet, P. (no affil.). *Bordeaux Med.* 8(6):625, 1975.

5666 QUANTITATIVE SCINTIGRAPHY OF THE LIVER AND PANCREAS. REVIEW. *(Fr.)* Desgrez, M. A. (no affil.). *Bordeaux Med.* 8(6):593, 1975.

5667 CORRELATIONS BETWEEN ENDOSCOPIC AND RADIOLOGIC FINDINGS FOR A DIAGNOSTIC ASSESSMENT OF DUODENAL BULB LESIONS. *(It.)* Cesaroni, R.; Mieli, C.; Nava, G. (S. Eugenio Hosp., Rome, Italy). *Minerva Gastroenterol.* 20(2):55, 1974.

5668 TECHNIQUE AND EVALUATION OF THE ^{198}Au-HEPATOGRAM. *(Ger.)* Seyss, R. (no affil.). *Med. Monatsschr.* 28(11):490-493, 1974.

5669 THE RELATION OF SERUM ENZYMOLOGY TO DIAGNOSIS OF HEPATIC METASTASIS. *(Fr.)* Kleisbauer, J. P.; Pietri, H.; Rosselo, R.; de Oliviera Lins, J. C.; Poirier, R.; Arnoux, A.; Laval, P. (Paoli-Calmette Inst., Marseille, France). *Arq. Gastroenterol.* 12(1):21-25, 1975.

5670 THE TREHALOSE-LOADING TEST IN MALABSORPTION SYNDROME. *(Ger.)* Dominick, A. C.; Anspach, K. (Child. Clin., Westphal. Univ. Munster, Germany). *Monatsschr. Kinderheilkd.* 123(5):432-433, 1975.

5671 THE SIGNIFICANCE OF COLONOSCOPY IN THE ESTIMATION OF DISEASES OF THE LARGE INTESTINE IN CHILDHOOD. *(Ger.)* Burdelski, M.; Lucking, T.; Seifert, E. (Med. Univ. Hannover, Germany). *Monatsschr. Kinderheilkd.* 123(5):434-435, 1975.

5672 SMALL INTESTINAL BIOPSY IN THE DIAGNOSIS OF INTESTINAL AND NON-INTESTINAL DISEASES. *(Sp.)* Cantor, D. S.; Costa, J. A. (Prof. Alejandro Posadas Polyclin., Haedo, Argentina). *Acta Gastroenterol. Latinoam.* 6(2):73-81, 1974.

5673 THE VALUE OF SCINTIGRAPHY IN THE DIAGNOSIS OF PANCREAS DISEASES. *(Ger.)* Muller-Brand, J. (Inst. Med. Radio., Univ. Basel, Switzerland). *Schweiz. Med. Wochenschr.* 105(21):675-678, 1975.

5674 STANDARD RECOMMENDATIONS FOR ORAL AND INTRAVENOUS ROENTGENOGRAPHIC STUDIES OF THE GALLBLADDER AND BILE DUCTS. *(Ger.)* Hasert, V.; Kroger, W.; Kunz, B.; Moldenhauer, W.; Preuss, H. J.; Richter, K.; Schmidt, G.; Moldenhauer, W. (Med. Radiol. Soc., Leipzig, Germany). *Radiol. Diagn. (Berl.)* 15(6):837-843, 1974.

5675 THE DIAGNOSTIC SIGNIFICANCE OF QUANTITATIVE ANALYSIS OF STOMACH SECRETION. *(Ger.)* Ewers, H. R.; Hengstebeck, W.; Brouwers, H. P. (Univ. Med. Clin., Essen, Germany). *Med. Welt* 26(12):533-540, 1975.

5676 DIAGNOSTIC AND THERAPEUTIC POSSIBILITIES FOR THE INTERNIST (INVOLVING THE PAPILLA OF VATER). *(Ger.)* Classen, M. (no affil.). *Fortschr. Med.* 93(5):197-199, 1975.

5677 ARTERIOGRAPHY OF Echinococcosis multilocularis (tyrolo-bavarian). *(Fr.)* Bessot, M.; Treheux, A.; Gaucher, P.; Regent, D.; Richaume, B.; Fays, J. (Cent. Hosp., Nancy, France). *Med. Chir. Dig.* 4(1):39-44, 1975.

5678 COMMENTS ON THE STANDARD RECOMMENDATIONS FOR ROENTGEN STUDIES OF THE GALLBLADDER AND BILE DUCTS. *(Ger.)* Moldenhauer, W.; Richter, K. (Med. Radiol. Soc., Rostock, Germany). *Radiol. Diagn. (Berl.)* 15(6):845-846, 1974.

5679 A MODIFICATION OF WELIN'S DOUBLE CONTRAST TECHNIQUE IN EXAMINATION OF THE COLON. *(Ger.)* Hauger, W. (Med. Radiol. Inst., Univ. Tubingen, Germany). *Roentgenpraxis* 28(1):1-5, 1975.

5680 RADIONUCLIDES IN THE DIAGNOSIS OF GASTROINTESTINAL DISEASE. *(Ger.)* Creutzig, H. (Med. Coll., Hannover, Germany). *Med. Welt.* 25(37):1473-1475, 1974.

5681 THE TECHNIQUE AND EVALUATION OF SECRETIN AND PANCREOZYMIN TESTS FOR EXOCRINE PANCREAS. *(Rus.)* Geller, L. I.; Kazimirchik, A. P.; Romanets, V. A.; Bulgakova, O. S. (Khabarovsk Med. Inst., USSR). *Lab. Delo* (4):214-218, 1975.

5682 THE USE OF RHEOHEPATOGRAPHY IN THE DIAGNOSIS OF LIVER PATHOLOGY. *(Rus.)* Reznik, N. D.; Vaisman, V. D. (Gorki Inst. Labour Hyg. Occupat. Diseases, USSR). *Ter. Arkh.* 46(12):111-114, 1974.

5683 TWO UNIQUE APPROACHES TO CHOLECYSTOGRAPHY. *(E.)* Lavery, J. P. (St. Vincent's Hosp., New York, N.Y.). *Am. J. Ther. Clin. Rep.* 1(1):67-73, 1975.

5684 INTRAOPERATIVE ESOPHAGOSCOPY, GASTROSCOPY, COLONOSCOPY, AND ENDOSCOPY OF THE SMALL BOWEL. *(E.)* Bombeck, C. T. (Abraham Lincoln Sch. Med., Chicago, Ill.). *Surg. Clin. North Am.* 55(1):135-142, 1975.

5685 CLINICAL SIGNIFICANCE OF GASTRIN RADIOIMMUNOASSAY. *(E.)* Walsh, J. H. (Dep. Med., Univ. California, Los Angeles). *Semin. Nucl. Med.* 5(3):247-254, 1975.

5686 THE VALUE OF THE DUODENAL INTUBATION METHOD (SELLINK MODIFICATION) FOR THE RADIOLOGICAL VISUALIZATION OF THE SMALL BOWEL. *(E.)* Fleckenstein, P.; Pedersen, G. (Rigshosp., Univ. Copenhagen, Denmark). *Scand. J. Gastroenterol.* 10(4):423-425, 1975.

5687 PREOPERATIVE DIAGNOSIS OF BLEEDING MECKEL'S DIVERTICULUM BY SCINTIGRAPHY. *(E.)* Steinberg, M.; Skipper, A.; Damsgaard-Sorensen, P. (Odense Univ. Hosp., Denmark). *Z. Kinderchir.* 16(1):93-96, 1975.

5688 ASSOCIATION OF AN ISOMERIC SPECIES OF CARCINOEMBRYONIC ANTIGEN WITH NEOPLASIA OF THE GASTROINTESTINAL TRACT. *(E.)* Edgington, T. S.; Astarita, R. W.; Plow, E. F. (Scripps Clin. Res. Found., La Jolla, Calif.). *New Engl. J. Med.* 293(3):103-107, 1975.

5689 RADIOLOGIC DIAGNOSIS OF PANCREATIC CANCER. *(E.)* Rosch, J. (Univ. Oregon Med. Sch., Portland). *J. Surg. Oncol.* 7(2):121-123, 1975.

5690 SECRETION ANALYSIS: SECRETIN TESTING IN PANCREATIC CANCER. *(E.)* Dreiling, D. A. (Mt. Sinai Sch. Med., New York, N.Y.). *J. Surg. Oncol.* 7(2):101-105, 1975.

5691 THE DIAGNOSIS OF MALABSORPTION IN THE TROPICS. *(E.)* Rowland, H. A. K. (London Sch. Hygiene Trop., Med., England). *Trop. Doct.* 5(1):3-7, 1975.

5692 ENDOSCOPIC EXAMINATION OF THE COLON AND RECTUM. *(E.)* Stearns, M. W. (Mem. Hosp. Cancer, New York, N.Y.). *S. Afr. Cancer Bull.* 19(1):28-31, 1975.

5693 RETROGRADE ENDOSCOPIC CHOLANGIO-PANCREATICOGRAPHY (ERCP). *(E.)* Myren, J. (Ulleval Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 10(3):229-230, 1975.

5694 LAPAROSCOPIC DIAGNOSIS OF THE CRUVEILHIER-BAUMGARTEN SYNDROME. COMMENTS ON THREE CASES, TWO OF THEM OCCULT. *(Sp.)* Herrerias, J. M.; Perez Mijares, R.; Marcias, A.; Jimenez, J.; Damiano, A.; Esteban, J.; Aljama, P.; Moreno, M. (Prof. Sch. Dig. Tract Dis., Cadiz, Spain). *Rev. Esp. Enferm. Appar. Dig.* 45(4):409-416, 1975.

5695 ENDOSCOPIC EVALUATION OF THE GASTRIC ANTRUM. *(E.)* Mann, N. S. (VA Hosp., Topeka, Kansas). *South. Med. J.* 68(5):541-542, 1975.

5696 THE RADIOLOGICAL DIAGNOSIS OF HEPATOMA WITH SPECIAL EMPHASIS ON ANGIOGRAPHY. *(E.)* Bass, E. M. (Groote Schuur Hosp., Cape Town, South Africa). *S. Afr. Med. J.* 49(18):745-751, 1975.

5697 FIBEROPTIC ENDOSCOPY: PRECISE DEFINITION OF UPPER GASTROINTESTINAL DISEASE. *(E.)* Knutson, C. O. (Univ. Louisville Sch. Med., Ky.). *Am. J. Surg.* 129(6):651-655, 1975.

5698 COLONOSCOPY: A REVIEW. *(E.)* Overholt, B. F. (St. Mary's Mem. Hosp., Knoxville, Tenn.). *Gastroenterology* 68(5):1308-1320, 1975.

5699 ANALYSIS OF LIVER SCANNING IN A GENERAL HOSPITAL. *(E.)* Oster, Z. H.; Larson, S. M.; Strauss, H. W.; Wagner, Jr., H. N. (Johns Hopkins Med. Inst., Baltimore, Md.). *J. Nucl. Med.* 16(6): 450-453, 1975.

5700 BILE CANALICULAR ANTIBODIES IN NON-HEPATIC DISEASES. *(E.)* Diederichsen, H. (Univ. Hosp., Odense, Denmark). *Lancet* (7916):1142-1143, 1975.

5701 STUDIES ON PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY: A SAFE AND EFFECTIVE TECHNIQUE BY LATERAL APPROACH. *(E.)* Lin, J. S. (Kumamoto Univ. Med. Sch., Japan). *Kumamoto Med. J.* 27(1):27-40, 1974.

5702 ECHOGRAPHIC STUDY OF THE PANCREAS. *(E.)* Leopold, G. R. (Univ. California, Sch. Med., San Diego). *JAMA* 232(3):287-289, 1975.

See also, 5705, 5726, 5890, 5920, 5931, 5937, 5966, 6006, 6024, 6047, 6076, 6083, 6118, 6166, 6181, 6183, 6210, 6224, 6263, 6350, 6362, 6368, 6375, 6383, 6392, 6398, 6434.

GASTROINTESTINAL DISEASES

ESOPHAGUS

5703 THE EPIDEMIOLOGY OF ESOPHAGEAL CANCER IN NORTH CHINA AND PRELIMINARY RESULTS IN THE INVESTIGATION OF ITS ETIOLOGICAL FACTORS. *(E.)* Anonymous. *Sci. Sinica* 18(1):131-148, 1975.

An epidemiological survey (involving 50 million people) of the incidence of esophageal cancer in North China was carried out from 1969 to 1971. Preliminary clinical and experimental studies were also carried out on etiological factors and associated conditions, and pathogenesis and histopathology of esophageal cancer. An area with a high mortality rate from esophageal cancer was located in the southern Taihang Mountains on the borders of the Honan, Shansi and Hopei provinces. The mortality rates decreased gradually, extending out from this area in irregular, concentric belts. The highest age-sex adjusted mortality rate reached 139.80/100,000 and the lowest was 1.43/100,000, with an average mortality rate of 37.39/100,000. The male:female patient ratio averaged 1.6:1. The higher the mortality rate of a locality, the lower the difference in the sex ratio. Over 60% of the total rate was in the age group ranging from 50 to 69 yr. In counties with a high incidence of esophageal cancer, there were also greater numbers of people with epithelial dysplasia of the esophagus. The average age-sex adjusted incidence rate in Lingsien county (1959-1970) was 108.56/100,000, and the mortality rate was 99.76/100,000. Mortality rate trends showed little change over 30 yr. The ratio of esophageal cancer to cancer of the gastric cardia was 3:1; most cases were located in the middle and lower third of the esophagus. A comparative study showed that in areas of high esophageal cancer incidence, gullet cancer among chickens was also common. It is concluded that there must be a close relation between this disease and the geographical environment.

5704 BENIGN EPITHELIAL HYPERPLASIA OF THE ESOPHAGUS: GLYCOGENIC ACANTHOSIS. *(E.)* Clemencon, G.; Gloor, F. (Canton Hosp., Olten, Switzerland). *Endoscopy* 6(4):214-217, 1974.

The epidemiology, morphology, and localization of glycogenic acanthosis of the esophagus in humans is discussed. Among 364 esophagogastroscopies performed between July 1973 and March 1974, glycogenic acanthosis of the esophagus was diagnosed in 24 patients (19 men and 5 women, average age, 64 yr). Fifteen of these patients had endoscopic signs of an incompetent cardia and 11 of these had clear endoscopic signs of reflux esophagitis. Biopsy specimens from 12 patients showed focal areas of thickened squamous epithelium with an increased number of rounded glycogen-rich squamous cells. There was no cellular atypia or dyskeratosis and no signs of inflammation. The lesions in glycogenic acanthosis consist of small, mostly multiple whitish plaques of raised hyperplastic epithelium; they are usually found on folds and can be stained dark brown or violet with Lugol's solution. Glycogenic acanthosis is often mistaken for leukoplakia on macroscopic examination, especially when the plaques are relatively large. The small plaques are missed on esophagoscopy when the lumen is not optimally distended by air insufflation or when using a very bright light. It is concluded that glycogenic acanthosis is a very frequent but harmless condition; although nothing is known of its etiology, it seems to be more frequent in cardiac incompetence.

5705 BARRETT'S ESOPHAGUS AND ADENOCARCINOMA. *(Fr.)* Savary, M.; Naef, A. P.; Ozzello, L.; Roethlisberger, B. (Otorhinolaryngol. Clin., Univ. Lausanne, Switzerland). *Schweiz. Med. Wochenschr.* 105(18):575-579, 1975.

Endoscopy was used to demonstrate various forms of cylindrical epithelial heterotopia with respect to frequency, pathological associations and relationship with adenocarcinoma. Of 6168 esophagoscopies performed between 1963 and 1974 (more than half for gastroesophaegeal reflux syndrome), 164 revealed the presence of single or multiple cylindrical epithelial zones. These zones were of two distinct types: 1) cylindrical epithelial islets of the upper (19%) or lower (4%) esophagus; 2) widespread and diffuse cylindrical eipthelial heterotopia of the lower esophagus (77%). The first type was a malformation of congenital origin without pathological significance. The second type, however, would seem to enter into the category of chronic peptic esophagitis and may represent a form of epithelial cicatrization of chronic peptic ulcers. It is concluded that adenocarcinoma (15%) of the lower esophagus is associated with the second type of cylindrical epithelial heterotopia. Treatment of peptic esophagitis is advisable before it reaches the chronic stage and, in particular, before the appearance of cylindrical epithelial zones.

5706 PATHOGENESIS AND TREATMENT OF REFLUX ESOPHAGITIS. *(It.)* Basile, A. (1st. Gen. Clin. Chir., Univ. Catania, Italy). *Chir. Gastroenterol.* 8(3):251-263, 1974.

5707 ACHALASIA OF CARDIA TREATED BY DILATATION. *(Ger.)* Sanatger, R.; Braun, L. (Chir. Clin., Univ. Munster, Germany). *Med. Klin.* 69(41): 1667-1671, 1974.

5708 STRANGULATED HIATAL HERNIA AND ITS TREATMENT WITH A CRINOPLATE. *(Fr.)* Lacaze, J. (no affil.). *Bordeaux Med.* 7(17):2555-2558, 1974.

5709 CANCER OF THE ESOPHAGUS AND GASTRIC CARDIA: DIAGNOSTIC PROBLEMS. *(Fr.)* Savary, M. (Otorhinolaryngblog. Clin., Univ. Lausanne, Switzerland). *Schweiz. Med. Wocheschr.* 105(18):550-555, 1975.

5710 MORPHOLOGICAL CHANGES OF ESOPHAGUS CANCER AFTER PREOPERATIVE RADIATION THERAPY. *(Rus.)* Gosh, T. E.; Pirogov, A. I.; Gritsman, Y. Y.; Papliyan, N. P.; Mamontov, A. S.; Konnova, L. V. (P. A. Gertsen Moscow Sci. Res. Inst. Oncol., USSR). *Med. Radiol. (Mosk.)* 19(7):21-26, 1974.

5711 EFFECT OF ANGIOARCHITECTURE OF THE SMALL INTESTINE ON CREATION OF AN ARTIFICIAL ESOPHAGUS. *(Rus.)* Dmitrienko, V. V.; Shraer, T. I. (Kemerovo Med. Inst., USSR). *Khirurgiia (Mosk.)* (12):66-71, 1974.

5712 THERAPY OF THE MALLORY-WEISS SYNDROME. *(Ger.)* Nauer, E.; Filippini, L. (Canton Hosp., Luzerne, Switzerland). *Dtsch. Med. Wochenschr.* 100(24):1343-1344, 1975.

5713 INDICATIONS AND LIMITS OF PRESSURE STUDIES IN THE EXPLORATION OF THE GASTRO-OESOPHAGEAL JUNCTION. *(It.)* Rovere, G. Q. D.; Farello, G. A.; Fracastoro, G.; Motton, G.; Perbellini, A.; Cordiano, C. (1st. Patol. Spec. Chir., Univ. Padova, Italy). *Acta Chir. Ital.* 30(3):253-271, 1974.

5714 OESOPHAGEAL STENOSIS FROM REFLUX. *(It.)* Rovere, G. Q. D.; Farello, G. A.; Motton, G.; Fracastoro, G.; Palma, F. D.; Corhiano, C. (1st. Patol. Spec. Chir., Univ. Padova, Italy). *Acta Chir. Ital.* 30(2):99-121, 1974.

5715 ABOUT THE RADIOTHERAPY OF TUMORS OF THE ESOPHAGUS. *(Ger.)* Wahlers, B.; Koppenfels, R. (Radiol. Klin., Wilhelms Univ., Munster, Germany). *Strahlentherapie* 149(3):252-262, 1975.

5716 SPONTANEOUS PNEUMOESOPHAGOGRAM. *(Ger.)* Dittrich, J. K.; Bautz, B. (Univ. Child. Clin., Marburg, Germany). *Fortschr. Rontgenstr.* 121(4):417-423, 1974.

5717 PRIMARY ADENOCARCINOMA OF THE ESOPHAGUS ARISING IN A COLUMNAR-LINED ESOPHAGUS. *(E.)* Stillman, A. E.; Selwyn, J. I. (Tucson Med. Cent., Ariz.). *Am. J. Dig. Dis.* 20(6):577-582, 1975.

5718 ESOPHAGEAL RECONSTRUCTION: AN EXPERIMENTAL APPROACH TO THE CONTROL OF REFLUX AFTER ESOPHAGEAL RESECTION. *(E.)* Henderson, R. D.; Fung, K.; Dube, P.; Marryatt, G. (Toronto Gen. Hosp., Canada). *Can. J. Surg.* 18(2):165-171, 1975.

5719 CONGENITAL ATRESIA OF THE OESOPHAGUS. A FOLLOW-UP STUDY IN 36 SURGICALLY CORRECTED CASES. *(E.)* Vaage, S.; Levorstad, K.; Efskind, L. (Univ. Hosp., Oslo, Norway). *Scand. J. Thorac. Cardiovasc. Surg.* 9(1):68-74, 1975.

5720 COLON ESOPHAGEAL BYPASS. *(E.)* Wilkins, Jr., E. W.; Burke, J. F. (Harvard Med. Sch., Boston, Mass.). *Am. J. Surg.* 129(4):394-400, 1975.

5721 REOPERATION AFTER ESOPHAGOMYOTOMY FOR ACHALASIA OF THE ESOPHAGUS. *(E.)* Ellis, Jr., F. H.; Gibb, S. P. (Lahey Clin. Found., Boston, Mass.). *Am. J. Surg.* 129(4):407-412, 1975.

5722 A RADIOLOGICAL STUDY OF 63 CASES OF OPERATED ESOPHAGEAL ATRESIA. *(E.)* Meradji, M.; Van Walleghem, J.; Vervat, D. (Sophia Child. Hosp., Rotterdam, Netherlands). *Radiol. Clin. Biol.* 43(6):559-570, 1974.

5723 ACQUISITION OF COLUMNAR (BARRETT TYPE) EPITHELIUM IN THE DISTAL ESOPHAGUS AFTER PARTIAL ESOPHAGOGASTRECTOMY. *(E.)* Hamilton, S. R.; Yardley, J. J. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *Lab. Invest.* 32(3):425-426, 1975.

5724 OESOPHAGEAL ATRESIA IN THE SOUTH WEST OF ENGLAND. *(E.)* David, T. J.; O'Callaghan, S. E. (Bristol Gen. Hosp., England). *J. Med. Genet.* 12(1):1-11, 1975.

5725 EVALUATION OF THE NISSEN FUNDOPLICATION FOR TREATMENT OF HIATAL HERNIA: USE OF PARIETAL CELL VAGOTOMY WITHOUT DRAINAGE AS AN ADJUNCTIVE PROCEDURE. *(E.)* Bahadorzadeh, K.; Jordan, P. H. (Baylor Coll. Med., Houston, Texas). *Ann. Surg.* 181(4): 402-408, 1975.

5726 BOERHAAVE SYNDROME: REPORT OF A CASE TREATED NON-OPERATIVELY. *(E.)* Larrieu, A. J.; Kieffer, R. (Univ. Md. Hosp., Baltimore). *Ann. Surg.* 181(4):452-454, 1975.

5727 CANCER OF THE OESOPHAGUS IN BRITTANY: AN INCIDENCE STUDY IN ILLE-ET-VILAINE. *(E.)* Tuyns, A. J.; Masse, G. (Int. Agency Res. Cancer, Lyon, France). *Int. J. Epidemiol.* 4(1):55-59, 1975.

5728 LOWER-ESOPHAGEAL WEBS. *(E.)* Tedesco, F. J.; Morton, W. J. (Washington Univ. Sch. Med., St. Louis, Mo.). *Am. J. Dig. Dis.* 20(4):381-383, 1975.

5729 SLIDING ESOPHAGEAL HIATUS HERNIA AND REFLUX PEPTIC ESOPHAGITIS. *(E.)* Woodward, E. R. (Dep. Surg., Univ. Fla., Gainesville). *Proc. Inst. Med. Chic.* 30(5):160-164, 1974.

5730 RADICAL SURGICAL TREATMENT OF ESOPHAGEAL STENOSIS DUE TO EPIDERMOLYSIS BULLOSA. *(E.)* Kralik, J.; Rapant, V. (Munic. Hosp., Olomouc, Czechoslovakia). *J. Thorac. Cardiovasc. Surg.* 69(5):790-792, 1975.

5731 PRIMARY ADENOID CYSTIC CARCINOMA OF THE ESOPHAGUS. REPORT OF A CASE AND REVIEW OF THE LITERATURE. *(E.)* Pourzand, A.; Freant, L.; Levin, R.; Peabody, J.; Absolon, K. (Washington Hosp. Cent., D.C.). *J. Thorac. Cardivasc. Surg.* 69(5):785-789, 1975.

5732 DOUBLE PRIMARY MALIGNANT LESION OF THE OESOPHAGUS--A CASE REPORT. *(E.)* Suri, R. K.; Singh, H.; Jain, S.; Gujral, J. S. (Post-Grad. Inst. Med. Educ. Res., Chandigarh, India). *Indian J. Cancer* 11(4):444-447, 1974.

5733 RADIOGRAPHIC FINDINGS IN ESOPHAGEAL ATRESIA WITH PROXIMAL POUCH FISTULA (TYPE B). *(E.)* Berdon, W. E.; Baker, D. H. (Dep. Radiol., Babies Hosp., New York, N.Y.). *Pediatr. Radiol.* 3(2):70-74, 1975.

See also, 5288, 5649, 5650, 5802, 5985, 6336, 6348, 6522, 6547.

STOMACH

5734 EFFECT OF UNILATERAL THORACIC VAGOTOMY ON HISTAMINE-STIMULATED ACID SECRETION IN MAN. *(E.)* Basso, N.; Coloni, G. F.; Ricci, C.; Passaro, Jr., E.; Speranza, V. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *J. Surg. Res.* 18(6):555-558, 1975.

The acid response to histamine stimulation in patients undergoing a thoracotomy for lung carcinoma was studied to determine the effect of unilateral vagotomy in man. Male thoracotomy patients (56, aged 37-62 yr) were studied pre- and postoperatively. There were five groups of patients: 1) vagus resection not necessary (32 patients); 2) vagotomy in the right diaphragm (five); 3) vagotomy at the right pulmonary hilum (five); 4) vagotomy at the left diaphragm (eight); and 5) vagotomy at the left pulmonary hilum (six). In all cases the acid response to a maximal dose of histamine (0.024 mg/kg) was measured before and after thoracotomy. The effect of unilateral vagotomy on gastric acid secretion depended on whether the right or left nerve was cut, and at what level the cut occurred. Resection of the left vagus at the level of the diaphragm reduced basal secretion and diminished acid response to histamine stimulation by 22%. No significant reduction in acid secretion was observed when vagal resection was done at the pulmonary hilum. Resection of the right vagus reduced basal secretion and the acid response to histamine stimulation by 45-50% when performed at either the pulmonary hilum or the diaphragm. These results may correlate with the observation that in man the right vagus is generally greater in size than the left. It is suggested that the right vagus is responsible for the major portion of the vagal innervation of the parietal cell mass and/or overlaps the innervation to the mucosa supplied by the left nerve. It is further concluded that unilateral right vagotomy in man may have therapeutic implications.

5735 METASTATIC INVOLVEMENT OF THE STOMACH SECONDARY TO BREAST CARCINOMA. *(E.)* Joffe, N. (Beth Israel Hosp., Boston, Mass.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(3):512-521, 1975.

Roentgenologic findings associated with gastric metastases secondary to breast carcinoma (based on nine such cases) are presented, including case reports of five of these women (51 to 81 yr). Autopsy in the first case revealed disseminated breast carcinoma with gross infiltration of the wall of the stomach (predominantly the muscularis) and submucosa by tumor cells identical to those seen in the primary breast lesions. Histological examination in the third case showed tumor infiltration of the serosa, muscularis and submucosa, with marked infiltration in the subserosal region of the stomach. An upper gastrointestinal examination in the fourth case revealed at least two large ulcer craters in the region of the narrowed prepyloric segment; however, no autopsy was obtained. Biopsy of the gastric mass in the fifth case revealed anaplastic carcinoma similar in appearance to the primary breast tumor. Based on the gross pathologic findings, two main types of roentgenologic abnormalities might be expected to occur: circumscribed nodular lesions or more diffuse, infiltrative lesions. The commonest type of roentgen abnormality was that produced by diffuse intramural infiltration of the stomach resulting in a linitis plastica appearance, with narrowing of the lumen secondary to gross thickening of the gastric wall, rigidity and diminished or absent peristalsis in the involved area. Roentgenologic studies of the second type of lesion may show single or multiple discrete intraluminal filling defects with or without associated ulceration. Because of the apparent rarity of this lesion, and until more information is available concerning the incidence of the discrete nodular form of gastric metastases from breast carcinoma, it is concluded that final diagnosis must depend upon histologic confirmation even in patients with disseminated disease.

5736 HISTOLOGIC TYPES AND POSSIBLE INITIAL STAGES IN EARLY GASTRIC CARCINOMA. *(E.)* Grundmann, E. (Inst. Pathol., Univ. Munster, Germany). *Beitr. Pathol.* 154(3):256-280, 1975.

In order to determine 1) which histologic types are found in early gastric cancer, 2) whether possible precursor and initial characteristic stages exist, and 3) from which proliferative zones these arise, 2003 gastric specimens from 301 patients were examined, and a diagnosis of carcinoma was made in 45 cases. Examination of resection preparations revealed 36 cases of deep invasive stomach carcinoma

and nine of early gastric cancer confined to mucosa and/or submucosa. Carcinomatous proliferations limited to mucosa or submucosa were classified in three histologic types: intestinal (adeno); mucocellular (signet ring cell); and anaplastic (solid). Mixed types were found combining the first and second, or the second and third types; one case presented a mixture of all three. Possible precursor or initial stages of all three types were found in a further 31 biopsies. Some were glandular lesions in superficial parts of the mucosa, previously described as possible preneoplastic stages of the intestinal type of early gastric cancer. "Signet ring cell drippings" from lower parts of tubule necks were recorded as an initial form of the signet ring cell type; this process is interpreted as detaching of isolated signet ring cells from a gland neck zone in progressing atypical transformation. An early neoplastic stage of the anaplastic type was identified in the gland neck dysplasia located exclusively in the antrum between surface mucosa and antrum glands, appearing rich in cells, and stretched. Early gastric cancer of this third type will arise in the same location. Conclusions from formal histogenesis suggest that the signet ring cell type and the anaplastic (solid) type might start in the lower part of the tubule necks. In consequence, the neck region of gastric glands could be the critical field of malignant transformation in gastric mucosa. It is concluded that long-term follow-up studies are needed to verify these observations and their interpretations.

5737 SURGICAL MANAGEMENT OF MENETRIER'S DISEASE WITH PROTEIN-LOSING GASTROPATHY. *(E.)*

Scott, Jr., H. W.; Shull, H. J.; Law, IV, D. H.; Burko, H.; Page, D. L. (Vanderbilt Univ. Med. Cent., Nashville, Tenn.). *Ann. Surg.* 181(5):765-777, 1975.

Case histories for three patients with Menetrier's disease and protein-losing gastropathy who were studied during a 12-yr period are presented. Characteristic findings which differentiate these patients from individuals with hypertrophic hypersecretory gastropathy, including the Zollinger-Ellison syndrome, were: 1) hypertrophy of gastric mucosa with giant rugal folds involving the fundus, cardia and body of the stomach but not the antrum; 2) mucosal hypertrophy consisting of gastric mucus-secreting cells while parietal and chief cells were diminished in number and were absent from many microscopic sections; 3) gastric secretion of large volume containing excess mucus, low to absent hydrochloric acid and protein concentrations five or six times the normal (1.7 mg/ml); 4) hypoalbuminemia and hypoglobulinemia due to loss of serum proteins from gastric mucosa into the gastric lumen; 5) rare association with gastric ulcer. Unlike patients with the Zollinger-Ellison syndrome, none of these had duodenal ulcer or multiple endocrine adenomatosis. Menetrier's disease with protein-losing gastropathy is a potentially lethal disorder of unknown cause and no specific treatment, although resection of the site of protein losses can be effective. One of the three patients presented here died before gastrectomy could be performed. The other two have remained well for 11 months and 12 yr, resp., after total gastrectomy with Roux-en-Y esophagojejunostomy and Hunt-Lawrence jejunal pouch. Because the manifestations of the disease have disappeared in these patients, and good rehabilitative results have been obtained, these procedures are recommended for patients with Menetrier's disease.

5738 THE EFFECT OF METOCLOPRAMIDE ON HUMAN GASTRIC EMPTYING AND SECRETION. *(E.)*

Meeroff, J. C. (Mayo Clin., Rochester, Minn.). *Acta Gastroenterol. Latinoam.* 6(2):55-61, 1974.

The effects of metoclopramide on gastric acid secretion and the rate of gastric emptying in normal subjects and duodenal ulcer patients were studied. Metoclopramide (10, 20, and 40 mg) was injected i.v. into 30 normal subjects, 30 duodenal ulcer patients with intact stomachs, and 30 duodenal ulcer patients who had undergone truncal vagotomy and pyloroplasty; in some cases histamine phosphate (0.04 mg/kg) was injected s.c. simultaneously with the metoclopramide. The effects on basal acid output, histamine-augmented acid output, the rate of gastric emptying of an isotonic saline "test meal" (500 ml infused into the stomach *via* gastric tube), and serum gastrin concentration were determined. Metoclopramide had no effect on basal output or histamine-stimulated acid output in the normal controls or duodenal ulcer patients. Although it significantly increased the rate of gastric emptying in the controls and duodenal ulcer patients who had undergone truncal vagotomy and pyloroplasty, metoclopramide had no effect on acid secretion or serum gastrin. The effects on gastric emptying were significantly greater with 40 mg metoclopramide than with 10 mg. In doses of 10-20 mg, metoclopramide had no side effects, but 40 mg caused mild to moderate watery diarrhea, abdominal spasms, and, in 60% of the patients, drowsiness. The results indicate that in humans metoclopramide increases the rate of gastric emptying in both intact and vagotomized stomachs, that this effect is not related to the release of gastrin, that the mechanism of action is independent of the integrity of the vagus nerves, and that metoclopramide has no effect on gastric acid secretion.

5739 BLOOD SEROTONIN AND GLUCOSE LEVELS IN PATIENTS WITH DUMPING SYNDROME AFTER ORAL LOADING WITH 100 ML OF 50% GLUCOSE. *(Pol.)*

Schabowski, J.; Pokora, J. (Inst. Intern. Dis., Med. Acad., Lublin, Poland). *Pol. Tyg. Lek.* 30(4):141-144, 1975.

In 24 patients with dumping syndrome (19 men and 5 women, aged 21-67 yr) blood serotonin and glucose levels, blood pressure and pulse rate were determined in the fasting state, and also 15 min, 30 min, and 1-5 hr after p.o. administration of 50% glucose. A control group of 9 patients (aged 32-62 yr) without dumping syndrome was subjected to similar treatment. All subjects in both groups had previously undergone partial gastrectomy. Four different types of behavior with regard to blood serotonin and glucose levels were observed in patients with dumping

syndrome: 11 patients (first type) showed a rise in serotonin concentration after the first 15 min with a considerable rise in glucose level followed by secondary hypoglycemia after 2-3 hr; 4 patients (second type) showed a rise in serotonin level after administration of glucose, while no significant changes occurred in the level of glucose; 5 patients (third type) showed a very high rise in glucose level followed by hypoglycemia after 2-3 hr, but without serotonin changes; and 4 patients (fourth type) showed no significant changes in serotonin and glucose levels as was also observed in controls.

5740 ROENTGENO-ENDOSCOPIC COMPARISONS IN DETERMINING THE PROXIMAL BOUNDARY OF CANCER OF THE STOMACH. *(Rus.)* Marmorshtein, S. Y.; Shnirelman, A. I.; Rozanov, Y. L.; Savinkin, Y. N. (P. A. Gertsen Moscow Sci. Res. Inst. Oncol., USSR). *Sov. Med.* (9):92-97, 1974.

5741 EMPLOYMENT OF QUATERON IN ASSOCIATION WITH PREDNISOLONE AND NEROBOL IN THE TREATMENT OF CHRONIC GASTRITIS WITH SECRETORY INSUFFICIENCY. *(Rus.)* Tsimmerman, Y. S.; Valirakhmanova, T. D. (Perm Med. Inst., USSR). *Vrach. Delo* (2):55-59, 1975.

5742 DIAGNOSTIC SIGNIFICANCE OF SALIVA ENZYMES IN GASTRIC DISEASES. *(Rus.)* Synovets, A. S.; Levitsky, A. P.; Osadchy, B. D. (Odessa Sci. Res. Inst. Stomatol., USSR). *Vrach. Delo* (2):61-63, 1975.

5743 COLIBACTERIN IN THE TREATMENT OF PATIENTS WITH CHRONIC GASTRITIS. *(Rus.)* Panchishina, M. V.; Vikhrova, F. K. (Lvov Med. Inst., USSR). *Vrach. Delo* (2):63-65, 1975.

5744 FUNCTIONAL AND MORPHOLOGICAL CHANGES OF THE STOMACH IN PATIENTS WITH TOXIC GOITER. *(Rus.)* Kotova, S. M. (Leningrad Sanit.-Hyg. Med. Inst., USSR). *Vrach. Delo* (1):25-28, 1975.

5745 SOME VARIANTS IN THE CLINICAL COURSE OF CANCER INVOLVING THE GASTRIC STUMP. *(Rus.)* Panchenko, K. P.; Bulgakov, G. A. (Inst. Gastroenterol., A. V. Vishnevskiy Inst. Surg. Acad. Med. Sci. USSR, USSR). *Sov. Med.* (2):37-40, 1975.

5746 GAMMA-THERAPY OF GASTRIC CARCINOMA. *(Rus.)* Sarkisyan, Y. K.; Garvey, N. N.; Kagan, Y. L. (Moscow Sci. Res. Inst. Radiography Radiol., Min. Public Health SSSR, USSR). *Med. Radiol. (Mosk.)* 19(7):26-29, 1974.

5747 MORPHOLOGICAL STATE OF THE GASTRIC MUCOSA IN THE CLINICAL PICTURE OF EXACERBATED CHRONIC GASTRITIS. *(Rus.)* Kanischev, P. A.; Pruglo, Y. V. (Dept. Gastric Dis., Dnepropetrovsk Sci. Res. Inst. Gastroenterol., USSR). *Sov. Med.* (2):16-21, 1975.

5748 GASTRIN-PRODUCING CELLS OF THE GASTRIC BODY MUCOSA IN SOME GASTRO-DUODENAL DISEASES. *(Rus.)* Uspensky, V. M. (Dept. Hosp. Ther., S. M. Kirov Military Med. Acad., Leningrad, USSR). *Vrach. Delo* (3):69-71, 1975.

5749 CONTRACTILE ACTIVITY OF THE GALLBLADDER AFTER VAGOTOMY IN CONNECTION WITH NON-RADICAL GASTRIC RESECTION. *(Rus.)* Postolov, P. M.; Stadkovich, V. S. (Clin. Surg., I. M. Sechenov First Moscow Med. Inst., USSR). *Khirurgiia (Mosk.)* (3):57-61, 1974.

5750 CLINICAL-CYTOLOGICAL PARALLELS IN PATIENTS WITH GASTRIC CANCER RECEIVING PRE-OPERATIVE GAMMA-THERAPY. *(Rus.)* Kolyadyuk, I. V.; Agamova, K. A. (P. A. Gertsen Moscow Sci. Res. Inst. Oncol., USSR). *Khirurgiia (Mosk.)* (4):70-76, 1974.

5751 GASTRIC FUNCTION BEFORE AND AFTER KIDNEY TRANSPLANTATION. STUDY BY PENTAGASTRIN TEST. *(Fr.)* Laplante, L.; Beaudry, C.; Thibert, F.; Dandavino, R. (Dept. Nephrology, Maisonneuve-Rosemont Hosp. Ctr., Univ. Montreal, Canada). *Union Med. Can.* 104(4):570-573, 1975.

5752 DETERMINATION OF GASTRIC EVACUATION BY MEANS OF GAMMA CAMERA. *(Dut.)* Dam, A. P. M. V.; Wagenmakers, G. (Med. Clin., St. Radboud Hosp., Nijmegen, Netherlands). *Tijdschr. Gastroenterol.* 17(2):87-93, 1974.

5753 PRIMARY LYMPHOSARCOMA OF THE STOMACH. *(It.)* Rasa, G.; Donati, A.; Morici, V.; Moscato, V. (1st. Chir. Anat., Univ. Catania, Italy). *Chir. Patol. Sper.* 22(2):98-113, 1974.

5754 ENDOSCOPIC FEATURES OF THE INCIPIENT LATE STAGE OF ACID-INDUCED CORROSIVE BURN OF THE STOMACH. *(Ger.)* Rohner, H. G.; Lelbach, W. K.; Muller, R. P.; Kuhr, J. (Med. Clin., Univ. Bonn, Germany). *Leber Magen Darm* 4(4):153-157, 1974.

5755 GASTRO-DUODENAL BAND AND DOUBLE PYLORUS. *(Ger.)* Pohl, W. (Med. Klin. III, Assoc. Hosp., Angsburg, Germany). *Leber Magen Darm* 4(4): 151-152, 1974.

5756 EARLY GASTRIC CANCER. *(Ger.)* Wesseler, T. (Radiol. Inst., Miners' Hosp., Recklinghausen, Germany). *Roentgenblaetter* 28(1):18-26, 1975.

5757 VASCULAR CHANGES IN GASTRIC CANCER WITH SPECIAL REFERENCE TO ANALYSIS OF AUTOPSY CASES WITHOUT HEMATOGENOUS METASTASES. *(Jpn.)* Nagao, K.; Matsuzaki, O.; Ide, G. (Chiba Univ. Med. Sch., Japan). *Gan No Rinsho* 1(2):103-109, 1975.

5758 PARTICIPATION OF THE BLOOD KININ SYSTEM IN DUMPING-REACTION IN PATIENTS WITH DUMPING SYNDROME. *(Rus.)* Kuzin, M. I.; Menshikov, V. V.; Chistova, M. A.; Dudnik, V. S.; Beliakov, N. V. (Clin. Surg., I. M. Sechenov First Moscow Med. Inst., USSR). *Khirurgiia (Mosk.)* (4):84-88, 1974.

5759 ABSORPTION OF VITAMIN B-12 AFTER GASTRIC RESECTION AND GASTRECTOMY. *(Rus.)* Goldin, V. A.; Appukhami, K. R. A.; Sakhibov, Y. D. (Clin. Gen. Surg., Moscow Med. Inst. Stomatol., USSR). *Khirurgiia (Mosk.)* (4):81-84, 1974.

5760 DIFFERENCES IN THE PATHOGENESIS OF SIMPLE ATROPHIC GASTRITIS AND ATROPHIC GASTRITIS IN PERNICIOUS ANEMIA. *(Cz.)* Fixa, B.; Komarkova, O.; Nozicka, Z. (Fac. Med., Charles Univ., Hradec Kralove, Czechoslovakia). *Vnitr. Lek.* 20(7): 638-642, 1974.

5761 AUTOIMMUNE PROCESSES IN PATIENTS WITH CHRONIC GASTRITIS. *(Rus.)* Kireeva, O. V.; Raskin, A. M.; Lisochkin, B. G. (S. M. Kirov Leningrad Inst. Postgrad. Training Phys., USSR). *Ter. Arkh.* 46(4):104-108, 1974.

5762 PULMONARY VARIANT OF THE COURSE OF GASTRIC CARCINOMA. *(Rus.)* Boiko, Y. G. (Dept. Pathol. Anat., Grodno Med. Inst., USSR). *Klin. Med. (Mosk.)* 53(3):69-73, 1975.

5763 CLINICO MORPHOLOGICAL CHARACTERISTICS OF GASTRIC LYMPHOMATOSIS. *(Rus.)* Tsodikov, G. V.; Smotrova, I. A.; Matveev, N. K.; Maiorov, V. M.; Vodolagin, V. D.; Ganchenko, L. I.; Gurvich, R. N.; Neugodov, V. A. (All-Union Sci. Res. Inst. Gastroenterol., Moscow, USSR). *Ter. Arkh.* 46(8):84-91, 1974.

5764 SIGNIFICANCE OF GASTROSCOPY IN DIAGNOSIS OF GASTRIC ULCERATIONS. *(Rus.)* Vodolagin, V. D.; Smolsky, B. G. (All-Union Sci. Res. Inst. Gastroenterol., Moscow, USSR). *Klin. Med. (Mosk.)* 52(11):52-55, 1974.

5765 ROENTGENOFIBROSCOPIC DIAGNOSIS AND TREATMENT OF BEZOARS OF THE STOMACH AND DUODENUM. *(Rus.)* Ovchinnikov, V. I.; Usova, N. A.; Rokhlin, E. A.; Pukhkaya, V. K. (Central Inst. Postgrad. Training Phys., S. P. Botkin Hosp., USSR). *Klin. Med. (Mosk.)* 52(7):83-88, 1974.

5766 BENIGN NON-EPITHELIAL TUMORS OF THE STOMACH. *(Rus.)* Azarova, I. V. (Clin. Surg., Leningrad Sanit.-Hyg. Med. Inst., USSR). *Vest. Khir.* 114(3):25-29, 1975.

5767 QUALITATIVE AND QUANTITATIVE HISTOENZYMOCHEMISTRY OF GASTRIC CANCER. *(Rus.)* Sklianskaia, O. A. (Dept. Path. Anat., I. M. Sechenov First Moscow Med. Inst., USSR). *Arkh. Patol.* 37(1): 38-45, 1975.

5768 GASTRO-COLONIC FISTULAE. *(Dan.)* La Cour Anderson, J. (Sundby Hosp., Kobenhaven, Denmark). *Ugeskr. Laeger* 137(7):375-377, 1975.

5769 THE EFFECT OF SYNTHETIC SALMON CALCITONIN (SMC) ON GASTRIC MOTILITY AND ACID SECRETION IN MAN. *(Ger.)* Paul, F.; Neumann, F.; Schmidt, F. W. (Med. Coll., Hannover, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:526-528, 1974.

5770 PREALBUMINS IN CANCEROUS TISSUE OF THE STOMACH. *(Rus.)* Lomachenkov, V. D. (Med. Inst., Smolensk, USSR). *Vopr. Onkol.* 21(2):32-34, 1975.

5771 CIRCULAR STENOSIS OF THE PYLORUS: MUSCULAR HYPERTROPHY OR CARCINOMA? *(Ger.)* Schirmer, G.; Brecht, T.; Koischwitz, D.; Schafer, P. K. (Univ. Radiol. Clin., Bonn, Germany). *Roentgenblaetter* 28(1):27-35, 1975.

5772 THE RESULTS OF JEJUNOGASTRIC RECONSTRUCTION IN DUMPING SYNDROME. *(Rus.)* Kradinov, A. I.; Volobuev, N. N.; Chirva, V. I. (Crimean Med. Inst., Simferopol, USSR). *Vestn. Khir.* 114(1):101-103, 1975.

5773 BENIGN NEUROGENIC TUMORS OF THE STOMACH. *(Rus.)* Azarova, I. V.; Kareva, A. I. (Leningrad Sanit.-Hyg. Med. Inst., USSR). *Vestn. Khir.* 114(2):22-26, 1975.

5774 THE ABSORPTION OF FAT AND GLUCOSE AFTER VAGOTOMY WITH NON-RADICAL RESECTION OF THE STOMACH. *(Rus.)* Postolov, P. M.; Fetisov, V. A.; Missura, N. V.; (1st Moscow Med. Inst., USSR). *Vestn. Khir.* 114(1):29-31, 1975.

5775 THE USE OF FIBEROPTICENDOSCOPE TO REMOVE POLYPS FROM THE STOMACH AND LARGE INTESTINE. *(Rus.)* Saveljev, V. S.; Buyanov, V. M.; Kornilov, Iu. M.; Balalykin, A. S. (2nd Moscow Med. Inst., USSR). *Vestn. Khir.* 114(2):26-31, 1975.

5776 GASTRIC PHLEGMON THAT DEVELOPED DURING CORTICOSTEROID TREATMENT FOR PSORIASIS. *(Pol.)* Dabrowski, Z.; Jelen, J.; Kardel-Mizerska, T. (Pomeranian Acad. Med., Szczecin, Poland). *Wiad. Lek.* 28(5):415-418, 1975.

5777 BENIGN TUMORS OF THE STOMACH. *(Fr.)* Rubay, J.; Barreau, J. (Saint-Joseph Hosp., Gilly, Belgium). *Acta Chir. Belg.* 74(1):25-44, 1975.

5778 CALCIFYING MUCINOUS ADENOCARCINOMA OF THE STOMACH--CASE REPORT AND DIFFERENTIAL DIAGNOSIS OF CALCIFYING PROCESSES IN THE UPPER ABDOMEN. *(Ger.)* Schubert, H. J.; Klein, H. J.; Christien, G. (Pathol. Inst., Univ. Koln, Germany). *Leber Magen Darm* 4(4):139-146, 1974.

5779 PYLORIC AND PREPYLORIC MUSCLE PLATES. *(Ger.)* Klein, H. J.; Schubert, H. J. (Pathol. Inst., Univ. Koln, Germany). *Leber Magen Darm* 4(4):147-150, 1974.

5780 ASSOCIATION OF MENETRIER'S DISEASE WITH GASTRIC POLYPOSIS, VILLOUS TUMOURS, AND GASTRIC CANCER: STUDY OF 5 CASES. *(Fr.)* Bourdais, J. P.; Hureau, J.; Delavierre, P. (Hosp. Vaugirard, Paris, France). *Ann. Gastroenterol. Hepatol.* 11(2):85-89, 1975.

5781 GLOMUS TUMOR OF THE STOMACH: AN ULTRASTRUCTURAL STUDY. *(E.)* Kanwar, Y. S.; Manaligod, J. R. (Abraham Lincoln Sch. Med., Univ. Illinois, Chicago). *Arch. Pathol.* 99(7):392-397, 1975.

5782 AN ANALYSIS OF TWENTY YEARS' EXPERIENCE WITH OPERATIONS FOR CARCINOMA OF THE STOMACH. *(E.)* White, IV., R. R.; Mackie, J. A.; Fitts, Jr., W. T. (Univ. Pennsylvania Med. Sch., Philadelphia). *Ann. Surg.* 181(5):611-615, 1975.

5783 PHYTOBEZOAR OCCURRING AS A COMPLICATION OF GASTRIC CARCINOMA. *(E.)* Van Thiel, D. H.; DeBelle, R. C.; Painter, T. D.; McMillan, W. B.; Haradin, A. R. (Dep. Med., Univ. Pittsburgh, Pa.). *Gastroenterology* 68(5):1292-1296, 1975.

5784 SYPHILITIC GASTROPATHY. *(E.)* Reisman, T. N.; Leverett, F. L.; Hudson, J. R.; Kalser, M. H. (Univ. Miami Med. Cent., Fla.). *Am. J. Dig. Dis.* 20(6):588-593, 1975.

5785 CLINIC AND COURSE OF EROSIONS OF THE GASTRIC MUCOSA. *(E.)* Krentz, K.; Gohrband, G. (no affil.). *Acta Hepatogastroenterol.* 22(1):65, 1975.

5786 SURGICAL RESULTS FOR EARLY GASTRIC CARCINOMA. ADITIONAL REPORT. *(E.)* Yamada, E. (Aichi Cancer Cent. Hosp., Nagoya, Japan). *Int. Surg.* 60(3):139-143, 1975.

5787 STRESS ULCERATION AND THE GASTRIC MUCOSAL BARRIER. *(E.)* Zinner, M. J.; Turtinen, L.; Gurll, N. J. (Walter Reed Army Inst. Res., Washington, D.C.). *Am. Surg.* 41(4):209-213, 1975.

5788 SCIRRHOUS CARCINOMA OCCURRING IN THE CORPUS (BODY) OF THE STOMACH. *(E.)* Nagayo, T.; Yokoyama, H. (Aichi Cancer Cent., Res. Inst., Nagoya, Japan). *Acta Pathol. Jpn.* 24(6):797-814, 1974.

5789 GASTRIC LEIOMYOBLASTOMA. *(E.)* Janvrin, S. B. (R. Berkshire Hosp., Reading, England). *Proc. Roy. Soc. Med.* 68(2):90, 1975.

5790 GASTROCOLIC FISTULA COMPLICATING BENIGN GASTRIC ULCER: CASE REPORT AND REVIEW OF LITERATURE. *(E.)* Taguchi, K.; Beaudoin, J. G. (R. Victoria Hosp., Montreal, Canada). *Can. J. Surg.* 18(2):186-189, 1975.

5791 INFANTILE HYPERTROPHIC PYLORIC STENOSIS IN BELFAST, 1957-1969. *(E.)* Dodge, J. A. (Welsh Nat. Sch. Med., Cardiff, Wales, U.K.). *Arch. Dis. Child.* 50(3):171-178, 1975.

5792 IS 'SECOND LOOK SURGERY' JUSTIFIED IN SUSPECTED RECURRENCES OF CANCER OF THE STOMACH? *(E.)* Ellis, H.; Jayasekara, G. (Westminster Hosp., London, England). *Br. J. Surg.* 62(3):226-230, 1975.

5793 A CASE OF HAEMORRHAGE FROM VOLVULUS OF THE GASTRIC FUNDUS. *(E.)* Metcalfe-Gibson, C. (Stepping Hill Hosp., Stockport, England). *Br. J. Surg.* 62(3):224-225, 1975.

5794 THE MANAGEMENT OF BLEEDING ACUTE GASTRIC EROSIONS: THE ROLE OF GASTRIC HYPOTHERMIA. *(E.)* Himal, H. S.; Watson, W. W.; Jones, C. W.; MacLean, L. D. (R. Victoria Hosp., Montreal, Canada). *Br. J. Surg.* 62(3):221-223, 1975.

5795 EXTRAMEDULLARY PLASMACYTOMA OF STOMACH. *(E.)* Habeshaw, J. A.; Hayward, M. J.; McVie, J. G. (Dep. Pathol., Univ. Edinburgh, Great Britain). *Scand. J. Haematol.* 14(1):57-64, 1975.

5796 STUDY OF GASTRIC CARCINOMA (377 CASES). *(E.)* Sharma, O. P. (Tata Mem. Hosp., Bombay, India). *Indian J. Cancer* 11(4):406-408, 1974.

5797 RECURRENT ULCER OF THE THORACIC STOMACH PENETRATING THE HEART: REPORT OF A CASE AND REVIEW OF THE LITERATURE. *(E.)* Lam, C. R.; Angulo, A. E.; Priest, R. J. (Henry Ford Hosp., Detroit, Mich.). *J. Thorac. Cardiovasc. Surg.* 69(5): 835-838, 1975.

5798 EFFECTS OF PHENTOLAMINE MESYLATE AND PROPRANOLOL HYDROCHLORIDE ON ASPIRIN-INDUCED GASTRIC LESIONS AND GASTRIC SECRETION IN THE RAT. *(E.)* Okabe, S.; Takeuchi, K.; Honda, K.; Takagi, K. (Dep. Chem. Pharmacol., Tokyo, Japan). *Jpn. J. Pharmacol.* 24(3):490-492, 1974.

See also, 5260, 5299, 5316, 5615, 5617, 5618, 5619, 5641, 5651, 5659, 5660, 5661, 5675, 5695, 5711, 5835, 5927, 5948, 5954, 6030, 6154, 6358, 6442, 6476, 6484, 6542.

5799 ELECTRICAL POTENTIAL DIFFERENCE, SODIUM ABSORPTION AND POTASSIUM SECRETION BY THE HUMAN RECTUM DURING CARBENOXOLONE THERAPY. *(E.)* Tomkins, A. M.; Edmonds, C. J. (Univ. Coll. Hosp., London, England). *Gut* 16(4):277-284, 1975.

The transmucosal electrical potential difference and sodium and potassium net fluxes were measured in patients on carbenoxolone therapy for peptic ulcer, and were compared to measurements in normal individuals. Of nine patients (aged 19-65 yr), six had gastric ulcer and three had duodenal ulcer; eight of these had no clinical evidence of disease. Each patient was studied before, during and after a 6-week course of carbenoxolone sodium (100 mg three times/day for 1 week, followed by 50 mg three times/day for 5 weeks). The ninth patient, a 62-yr-old man with gastric ulcer, developed fluid retention while on the drug, but remained in the experiment while in the hospital. Potential difference was measured with probe and skin electrodes. Flux measurements were obtained with dialysis tubes mounted on rubber catheters and containing 1.6 ml solution. There was a rise in transmucosal potential difference persisting throughout treatment in all subjects, accompanied by an increase in sodium absorption and potassium secretion. Comparison of the potential difference changes produced by carbenoxolone with those due to the mineralocorticoid 9-α-fluorocortisol (taken by the two controls) showed that carbenoxolone had about 0.001% the potency on a weight basis, and the two drugs appeared to be additive in their effects. Topical instillation of carbenoxolone produced an elevation of potential difference which persisted for 3 days. Amiloride and bendrofluazide did not interfere with these actions of carbenoxolone, but spironolactone abolished them. The patient with fluid retention and hypokalemia had a rectal potential difference similar to that of the other patients who had no side effects. It is concluded that carbenoxolone consistently produces mineralocorticoid activity in the rectal mucosa of all individuals taking the drug, and that this may reflect an inherent property of the drug.

5800 THE EFFECT OF INTRAVENOUS INFUSIONS OF PROSTAGLANDINS E_2 AND $F_{2\alpha}$ ON HUMAN GASTRIC FUNCTION. *(E.)* Newman, A.; de Moraes-Filho, J. P. P.; Philippakos, D.; Misiewicz, J. J. (Cent. Middlesex Hosp., London, England). *Gut* 16(4):272-276, 1975.

The effect of i.v. infusions of prostaglandins E_2 and $F_{2\alpha}$ at various dose levels on basal or on maximally or submaximally pentagastrin-stimulated acid secretion was studied in 40 male subjects with peptic ulcer, x-ray negative dyspepsia, or no gastrointestinal disease. Each received a continuous i.v. infusion of either 0.15 M saline, pentagastrin or prostaglandins. Prostaglandin $F_{2\alpha}$ (0.08 μg/kg/min) transiently, but significantly, inhibited submaximal acid output and increased the frequency of antral contractions. Prostaglandin E_2 (0.08 μg/kg/min) inhibited basal acid secretion. It is concluded that the inhibition of acid secretion by prostaglandin $F_{2\alpha}$ is of interest in emphasizing the species differences in responses to this agent. Further, additional studies of antral contractibility in individuals with more active antral contractions are indicated, since in these persons, only infrequent contractions were observed in the basal state, averaging 1/min.

5801 THE EFFECT OF DIAZEPAM ON PENTAGASTRIN-STIMULATED AND NOCTURNAL (SLEEPING) GASTRIC SECRETION IN MAN. *(E.)* Roberts, D. M.; Oldrey, T. B. N. (R. Army Med. Coll., London, England). *Am. J. Gastroenterol.* 63(5):396-399, 1975.

The effects of diazepam on stimulated gastric secretion and on unstimulated nocturnal secretion during sleep were studied in 10 male patients with dyspepsia (six with duodenal ulcer). Gastric secretion was measured for 3 hr following an s.c. injection of pentagastrin (6 μg/kg), an i.v. injection of diazepam (10 mg), and/or an i.v. injection of atropine (1.2 mg). Gastric secretion was also measured for 7 hr during confirmed nocturnal sleep that was preceded in some cases by an i.v. injection of diazepam (10 mg). The mean acid output after pentagastrin alone was 25.96, 13.00, and 5.61 mM HCl in the 1st, 2nd, and 3rd hr, resp. The addition of diazepam to pentagastrin resulted in a significant reduction in acid output during the 2nd (52.6% reduction) and 3rd (75.0% reduction) hr. The further addition of atropine resulted in a further reduction of 31.4% in the 1st hr, 42.0% in the 2nd hr, and 76.4% in the 3rd hr. The effects of diazepam and atropine on the pentagastrin-induced volume output were similar. The mean unstimulated nocturnal acid secretion over 7 hr was 24.83 mM HCl and the mean volume output was 338 ml. Diazepam did not significantly alter these parameters. The results suggest that diazepam acts during wakefulness by inhibiting secretory stimuli arising centrally, thus providing a rationale for use of this drug in patients with peptic ulcer in whom stress may be a pathogenic factor.

5802 GASTRO-OESOPHAGEAL REFLUX COMPLICATING HIGHLY SELECTIVE VAGOTOMY. *(E.)* Temple, J. G.; McFarland, J. (R. South. Hosp., Liverpool, England). *Br. Med. J.* 2(5964):168-169, 1975.

Patients with chronic duodenal ulceration and no evidence of pyloric stenosis, gastroesophageal reflux, or hiatus hernia underwent highly selective vagotomy. Of 20 patients, five (three men and two women, aged 41-56 yr) developed persistent symptoms of gastroesophageal reflux, with the usual features of heartburn, retrosternal discomfort and regurgitation accompanied by increased flatulence. This complication developed shortly after surgery in three patients and between 6 and 12 months in the remaining two. Postoperative barium meal examination showed gastroesophageal reflux in four patients,

with evidence of hiatus hernia in three. All these patients showed normal gastric emptying after the barium meal. All required constant medication for their discomfort and all were classified as grade III on the Visick scale. The insulin test was positive in four of these patients. It is suggested that as a result of the greater mobilization of the stomach which is necessary in highly selective vagotomy, a degree of gravitational dependence develops which may alter slightly the angle of entry of the esophagus into the stomach. Since making the observations in these five cases, a practice has been instituted of narrowing the angle of His by opposing the stomach to the esophagus with a continuous chromic catgut stitch run up for about 5 cm from the esophagogastric junction. This simple procedure may readily be added when small vagal fibers are being cleared from the area.

5803 EFFECT OF THERAPEUTIC DOSES OF ULTRASOUND ON VASCULAR PERMEABILITY OF THE GASTRIC MUSOSA IN PATIENTS WITH PEPTIC ULCERS. *(Rus.)* Nikitin, G. A. (Smolensk Med. Inst., USSR). *Sov. Med.* (2):53-56, 1975.

5804 EFFECT OF SELECTIVE PROXIMAL VAGOTOMY WITH AND WITHOUT PYLOROPLASTY UPON BASAL AND FOOD STIMULATED SERUM IMMUNOREACTIVE GASTRIN IN PATIENTS WITH DUODENAL ULCER. *(Ger.)* Bauer, H.; Arnold, R.; Track, N. S.; Creutzfeldt, W.; Holle, F. (Med. Univ. Gottingen, Germany). *Klin. Wochenschr.* 53(5):209-214, 1975.

5805 THERAPY OF GASTRIC AND DUODENAL ULCERS. CLINICAL-STATISTICAL INVESTIGATION ON THE USE OF A NEW DRUG: SULPHOGLYCOPEPTIDE. *(It.)* Fazzini, G.; Giampaglia, G.; Scarpulla, P. (Civil Hosp., Padua, Italy). *Minerva Chir.* 29(17):933-935, 1974.

5806 EXOCRINE PANCREATIC FUNCTION IN PEPTIC ULCER PATIENTS LONG AFTER GASTRECTOMY. *(Rus.)* Zinevich, V. P. (Leningrad Inst. Grad. Med. Studies, USSR). *Vestn. Khir.* 114(2):31-34, 1975.

5807 SURGICAL ASPECTS OF THE ACIDOGENIC FUNCTION OF THE STOMACH IN PATIENTS WITH PEPTIC ULCER. *(Rus.)* Umbrumyants, O. A. (2nd Moscow Med. Inst., USSR). *Vestn. Khir.* 114(2):12-16, 1975.

5808 ETIOPATHOGENESIS OF PEPTIC ULCER. *(Ser.)* Zhivanovich, M.; Svirchevich, A.; Vukovich, V. (Novi Sad Hosp., Yugoslavia). *Med. Pregl.* 27(7-8):321-330, 1974.

5809 THE CLINICAL SIGNIFICANCE OF EXAMINATION OF GASTRIC SECRETION BY MAXIMAL STIMULATION WITH HISTAMINE IN PATIENTS WITH DUODENAL ULCER. *(Cz.)* Jablonska, M.; Fucik, M.; Souckova, E.; Prazak, J. (Fac. Gen. Med., Charles Univ., Prague, Czechoslovakia). *Vnitr. Lek.* 20(3):241-250, 1974.

5810 GASTROINTESTINAL BLEEDING AFTER GASTRECTOMY FOR PEPTIC ULCER. *(Rus.)* Abushakhmanov, Kh. A. (Karaganda Med. Inst., USSR). *Vestn. Khir.* 114(1):126-130, 1975.

5811 CHRONIC PEPTIC ULCERS IN CHILDREN AND ADOLESCENTS. *(Pol.)* Rondio, H.; Wierzbolowicz, J.; Sowinska, J. (Munic. Child. Hosp., Warsaw, Poland). *Pediatr. Pol.* 50(2):149-159, 1975.

5812 TITRATION CURVES FOR GASTRIC SECRETION: A STUDY ON DUODENAL ULCER AND GASTRIC ULCER WITH PARTICULAR REFERENCE TO THE EFFECT OF GLYCOPYRRONIUM. *(Dan.)* Kristensen, M. (no affil.). *Ugeskr. Laeger* 136(51):2877-2878, 1974.

5813 PHYTOBEZOAR AND PEPTIC ULCER. *(Rus.)* Iusubov, D. R. (1st Railroad Hosp., Donetsk, USSR). *Vestn. Khir.* 114(4):116, 1975.

5814 COPROLOGICAL STUDIES OF PATIENTS WITH PEPTIC ULCER BEFORE AND AFTER SURGERY. *(Rus.)* Sedasheva, Ia. E. (Kharkov Res. Inst. Gen. Emergency Surg., USSR). *Lab. Delo.* (4):246, 1975.

5815 STUDY OF THE LATE RESULTS OF 702 TOTAL VAGOTOMIES FOR DUODENAL ULCER. *(Fr.)* Hollender, L. F.; Bur, F.; Otteni, F.; Alexiou, D. (Serv. Gen. Surg., Strasbourg, France). *Chirurgie* 100(11):795-805, 1974.

5816 LESIONS OF THE PANCREAS, LIVER AND BILE OUTFLOW DUCTS IN PATIENTS WITH ULCEROUS DISEASE. *(Rus.)* Klimov, V. N.; Permiakov, (Dept. Clin. Surg., Sverdlovsk Med. Inst., USSR). *Vestn. Khir.* 114(3):21-25, 1975.

5817 SELECTIVE PROXIMAL VAGOTOMY WITHOUT GASTRIC DRAINAGE IN THE MANAGEMENT OF DUODENAL ULCER. *(Rus.)* Kuzin, M. I.; Postolov, P. M. (Fac. Surg. Clin., I. M. Sechenov First Moscow Med. Inst., USSR). *Khirurgiia (Mosk.)* (12):38-44, 1974.

5818 EXPERIENCE WITH BISPAN IN ULCER DISEASE. *(Rus.)* Galetskaia, T. M.; Mironova, K. A.; Okhonko, V. I.; Spivak, O. M.; Fomina, A. A.; Liashko, N. A. (Kiev Med. Inst., USSR). *Vrach. Delo* (1):15-18, 1975.

5819 UROPEPSINOGEN AND GASTRIC SECRETION IN DUODENAL ULCER. *(Rus.)* Davidenko, L. A.; Udalov, I. A. (no affil.). *Vrach. Delo* (1):18-19, 1975.

5820 BILE SECRETION AND THE FUNCTIONAL STATE OF THE BILIARY SYSTEM IN PATIENTS WITH ULCER DISEASE. *(Rus.)* Okhrimovich, L. M. (Zeleshchiki Central District Hosp., Ternopol Region, USSR). *Vrach. Delo* (1):19-22, 1975.

5821 OUR EXPERIENCE OF EMPLOYMENT OF CHOLINERGIC BLOCKING AGENTS IN THE TREATMENT OF ULCER DISEASE. *(Rus.)* Burchinskii, G. I.; Galetskaia, T. M.; Ivanova, E. A.; Kaskevich, L. M.; Litinskaia, E. V.; Reinhardt, B. K.; Spivak, A. M. (Kiev Med. Inst., USSR). *Vrach. Delo* (2):70-76, 1975.

5822 SUBSTANTIATION OF THE REFLUX THEORY FOR PATHOGENESIS OF GASTRODUODENAL ULCER. *(Rus.)* Viteskii, Y. D. (no affil.). *Sov. Med.* (9):86-92, 1974.

5823 MEASUREMENT OF THE GASTRIC EVACUATION TIME BY MEANS OF RADIOACTIVE ISOTOPES AND ITS USE IN SURGERY FOR GASTRODUODENAL ULCER. *(Ger.)* Meves, M.; Beger, H. G.; Braun, D. (Surg. Klin., Free Univ. Berlin, Germany). *Chirurg* 45(10):459-462, 1974.

5824 THE HOLLANDER TEST: UNSUITABLE METHOD FOR THE ASSESSMENT OF THE RESULT AFTER PROXIMAL SELECTIVE VAGOTOMY. *(Ger.)* Largiader, F.; Sauberli, H.; Deyhle, P.; Jenny, S.; Nuesch, H. J.; Vetter, W.; Ammann, R. (Kanton Hosp., Zurich, Switzerland). *Schweiz. Med. Wochenschr.* 105(14):430-432, 1975.

5825 ACIDIMETRIC STUDY IN PATIENTS WITH DUODENAL ULCER. *(Bul.)* Gelov, N. (Principal Regional Hosp., Khaskovo, Bulgaria). *Khirurgiia (Sofiia)* 27(4):292-295, 1974.

5826 DUODENOFIBROSCOPY IN THE STUDY OF DUODENAL ULCER. *(Rus.)* Brailsky, K.; Popov, P.; Velev, G.; Chekhlarov, V. (Inst. Gastroenterol., Med. Acad., Sofia, Bulgaria). *Klin. Med. (Mosk.)* 52(10):102-105, 1974.

5827 DIAGNOSIS OF GASTRODUODENAL ULCERS. *(Rus.)* Khazanov, A. I.; Kalinin, A. V.; Barchunov, B. N.; Dmitriev, G. I.; Salvitskii, B. N.; Verteletskii, V. V. (N. N. Burdenko Princ. Mil. Hosp., USSR). *Klin. Med. (Mosk.)* 52(1):110-113, 1974.

5828 GASTROFIBROSCOPIC PECULIARITIES IN COMPLICATED PEPTIC ULCER. *(Rus.)* Samson, E. I.; Gaidichuk, S. T.; Malyukh, L. S. (Dept. Clin. Ther., Chernovtsy Med. Inst., USSR). *Klin. Med. (Mosk.)* 52(1):96-100, 1974.

5829 PARTIAL GASTRIC SECRETION IN PATIENTS WITH PEPTIC ULCER UNDER THE EFFECT OF TREATMENT WITH FILATOV'S SERUM AND BENZOHEXONIUM. *(Rus.)* Petrova, A. N.; Dzharimov, A. Y. (Krasnaya Armiya Kuban Med. Inst., Krasnodar, USSR). *Klin. Med. (Mosk.)* 52(11):55-58, 1974.

5830 SOME REGULARITIES OF HEMODYNAMIC CHANGES IN ULCER DISEASE. *(Rus.)* Nikitin, G. A. (Smolensk Med. Inst., USSR). *Vrach. Delo* (3):48-52, 1975.

5831 HEARTBURN IN ASSOCIATION WITH HIGH GASTRIC ACIDITY AS AN EARLY SYMPTOM OF ULCER DISEASE. *(Rus.)* Solovei, M. G. (S. P. Botkina Hosp., Moscow, USSR). *Vrach. Delo* (2):68-69, 1975.

5832 EFFECT OF AN OXYGEN COCKTAIL ON THE COURSE OF ULCER DISEASE. *(Rus.)* Stetsenko, G. S. (Izyaslav Cent. Dis. Hosp., USSR). *Vrach. Delo* (3):63-65, 1975.

5833 A TRIAL OF EVALUATION OF THE FREQUENCY OF COEXISTENCE OF GASTRIC AND DUODENAL ULCER WITH PANCREATIC DISEASE. *(Pol.)* Chylinski, J.; Adrich, Z. (Inst. Internal Dis., Med. Acad., Gdansk, Poland). *Wiad. Lek.* 28(3):177-180, 1975.

5834 THERAPEUTIC VALUE OF SODIUM BROMIDE AND POLOCAINE IN PEPTIC ULCER. *(Pol.)* Oleksy, J. (Reg. Mil. Hosp., Cracow, Poland). *Przegl. Lek.* 31(10):379-382, 1974.

5835 JUXTA-CARDIAL DIVERTICULAR DISEASE AND PEPTIC ULCER OF THE GASTRIC FUNDUS. *(It.)* Teixeira, P. A. (Dept. Surg., Antonio Luiz Sayao Sanat., Araras, Brazil). *Chir. Gastroenterol.* 7(2):200-205, 1973.

5836 RADIOLOGICAL APPEARANCE OF UPPER DIGESTIVE TRACT AFTER VAGOTOMY AND PYLOROPLASTY. *(Pol.)* Raszke, K.; Domanski, Z.; Zalewski, H. (Radiol. Dept., Med. Acad., Szczecin, Poland). *Pol. Przegl. Radiol.* 38(5):593-600, 1974.

5837 SOME INDICES OF THE FUNCTIONAL STATE OF THE HEART IN PATIENTS WITH ULCER DISEASE. *(Rus.)* Gurevich-Rabkina, F. P. (Kalinin First Munic. Hosp., Vitebsk, USSR). *Vrach. Delo* (1):22-25, 1975.

5838 MECHANISM OF ANTACID-INDUCED RELEASE OF GASTRIN. *(Ger.)* Feurle, G. (Med. Polyclin., Univ. Heidelberg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:515-516, 1974.

5839 GASTRIN AND DUODENAL ULCERS. *(Ger.)* Arnold, R.; Creutzfeldt, C.; Track, N. S.; Creutzfeldt, W. (Med. Clin., Univ. Gottingen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:368-377, 1974.

5840 THE THERAPEUTIC VALUE OF ASCORBIC ACID BEFORE AND AFTER SURGERY IN PATIENTS WITH COMPLICATED PEPTIC ULCER. *(Rus.)* Mordvinkina, T. N. (No affil.). *Vestn. Khir.* 114(1):143-145, 1975.

5841 TREATMENT OF PEPTIC ULCER WITH VAGOTOMY AND PYLOROPLASTY OR VON HABERER-SPATH'S ANTRECTOMY PROCEDURE. *(Ger.)* Tzamalukas, G.; Jakowidis, T. (Dresden-Neustadt Hosp., Germany). *Zentrabl. Chir.* 99(49):1544-1548, 1974.

5842 THE THERAPEUTIC EFFECTS OF UROGASTRONE ON GASTRIC ULCERS: THESE EFFECTS CAN BE ATTRIBUTED PRIMARILY TO ITS INHIBITORY ACTION ON HYPOTHALAMIC CENTERS REGULATING GASTRIC SECRETION AND MOTILITY. *(Fr.)* La Barre, J. (Fac. Med., Free Univ. Brussels, Belgium). *Brux. Med.* 54(12):687-692, 1974.

5843 TREATMENT OF PEPTIC ULCER WITH A STANDARD EXTRACT FROM Centella asiatica. *(Fr.)* Pergola, F. (Fac. Cochin, Port-Royal, France). *Med. Chir. Dig.* 3(6):445-448, 1974.

5844 CAUSES OF POSTOPERATIVE COMPLICATIONS AND DEATH IN PATIENTS WITH PEPTIC ULCER. *(Rus.)* Gadzhiev, A. S.; Ananev, V. G.; Spivak, V. P.; Ruban, V. M. (Dnepropetrovsk Res. Inst. Gastroenterol., USSR). *Klin. Khir.* (4):19-24, 1975.

5845 ASPECTS OF SURGICAL POLICY AND TECHNIQUES IN TREATMENT OF GASTRIC AND DUODENAL ULCERS WITH RADICAL SURGERY. *(Rus.)* Matiashin, I. M.; Baltaytis, Iu. V.; Boterashvili, M. I. (Kiev Med. Inst., USSR). *Klin. Khir.* (4):1-6, 1975.

5846 SURGICAL TREATMENT OF COMPLICATIONS FROM CHRONIC GASTRIC AND DUODENAL ULCERS. *(Rus.)* Napalkov, P. N.; Trunin, M. A.; Plotnikov, Iu. V. (Leningrad Med. Inst. Sanit. Hyg., USSR). *Klin. Khir.* (4):12-16, 1975.

5847 THE COURSE OF GASTRIC ULCER ASSOCIATED WITH PANCREATIC ADENOMA AND HETEROTOPIA OF THE ACCESSORY LOBE OF THE PANCREAS. *(Rus.)* Nechiporuk, V. M.; Rizhanovskaia, V. M. (N. I. Pirogov. Med. Inst., Vinnitsa, USSR). *Vestn. Khir.* 114(2): 125-129, 1975.

5848 PEPSIN SECRETION IN CONTROL SUBJECTS AND PATIENTS WITH DUODENAL ULCER. *(E.)* Desai, H. G.; Zaveri, M. P.; Salvi, D. P.; Antia, F. P. (BYL Nair Charitable Hosp., Bombay, India). *Indian J. Med. Res.* 63(2):278-281, 1975.

5849 PARIETAL CELL VAGOTOMY--ADVANTAGES AND POSSIBLE DISADVANTAGES. *(E.)* Amdruf, E. (Aarhus Kommunehospital, Denmark). *Bull. Soc. Int. Chir.* 33(5-6):396-404, 1975.

5850 ONE HUNDRED PATIENTS FIVE YEARS AFTER SELECTIVE GASTRIC VAGOTOMY AND DRAINAGE FOR DUODENAL ULCER. *(E.)* Jensen, H. E.; Strand, L.; Amdrup, E. (Kommunehospitalet, Copenhagen, Denmark). *Bull. Soc. Int. Chir.* 33(5-6):411-416, 1975.

5851 SELECTIVE GASTRIC VAGOTOMY RANDOMIZED WITH ANTRECTOMY AND PYLOROPLASTY: A PROSPECTIVE STUDY. *(E.)* Sawyers, J. L.; Scott, Jr., H. W. (Vanderbilt Univ. Sch. Med., Nashville, Tenn.). *Bull. Soc. Int. Chir.* 33(5-6):426-430, 1975.

5852 PATHOGENESIS AND AETIOLOGY OF ULCER DISEASE. PART II: DUODENAL ULCER. *(E.)* Blum, A. L.; Peter, P.; Krejs, G. J. (Med. Clin., Stadtspitals Triemli, Zurich, Switzerland). *Acta Hepatogastroenterol.* 22(2):123-218, 1975.

5853 EMERGENCY VAGOTOMY WITH A DRAINAGE PROCEDURE FOR PERFORATED GASTRIC AND DUODENAL ULCER: STUDY OF 73 CASES. *(E.)* Vardoulis, J.; Stenos, J.; Avgoustis, A.; Lefkidis, C. (Gen. Hosp., Volos, Greece). *Int. Surg.* 60(4):226-228, 1975.

5854 A PATTERN OF SERUM AMYLASE CONCENTRATION IN THE INITIAL 24 HOURS AFTER PERFORATION OF DUODENAL ULCERS. *(E.)* Imrie, C. W.; Whyte, A. S.; Frew, E. M. S. (R. Infirm., Glasgow, Scotland). *J. R. Coll. Surg. Edinb.* 19(6):370-373, 1974.

5855 THE EFFECT OF PYLOROPLASTY ON THE SIZE OF GASTRIC OUTLET. *(E.)* Taylor, T. V.; Hegarty, M. M.; Davies, M.; Torrance, H. B. (R. Infirm., Manchester, England). *J. R. Coll. Surg. Edinb.)* 19(5):292-296, 1974.

5856 MANAGEMENT OF THE RECURRENT ULCER. *(E.)* Lindenauer, S. M.; Dent, T. L. (Dep. Surg., Univ. Mich., Ann Arbor). *Arch. Surg.* 110(5):531-536, 1975.

5857 ACUTE PERFORATED DUODENAL ULCER. AN EVALUATION OF SURGICAL MANAGEMENT. *(E.)* Sawyers, J. L.; Herrington, Jr., J. L.; Mulherin, Jr., J. L.; Whitehead, W. A.; Mody, B.; Marsh, J. (Vanderbilt Univ. Sch. Med., Nashville, Tenn.). *Arch. Surg.* 110(5):527-530, 1975.

5858 GASTRIC ULCER CONTROLLED TRIAL USING A LICORICE DERIVATIVE RUCEDAL. *(E.)* Scobie, B. A. (Wellington Hosp., N.Z.). *N.Z. Med. J.* 81(536): 298-299, 1975.

5859 THE CAUSE AND TREATMENT OF POSTVAGOTOMY DIARRHOEA. *(E.)* Condon, J. R.; Robinson, V.; Suleman, M. I.; Fan, V. S.; McKeown, M. D. (St. Mary's Hosp., Eastbourne, England). *Br. J. Surg.* 62(4):309-312, 1975.

See also, 5216, 5314, 5617, 5742, 5875, 6048, 6521.

5860 MECKEL'S DIVERTICULUM. SHOULD IT BE EXCISED INCIDENTALLY AT OPERATION? *(E.)*
Michas, C. A.; Cohen, S. E.; Wolfman, Jr., E. F. (Dep. Surg., Univ. California, Davis). *Am. J. Surg.* 129(6):682-685, 1975.

During a 7-yr period, 35 cases of Meckel's diverticulum were recorded, 33 discovered at operation and two at autopsy. Of the 33 found at operation, 21 were found incidentally during surgery for other diseases, and 12 represented the primary source of the signs or symptoms for which celiotomy was performed. In the series presented here, there were 18 males and 17 females (aged 7 weeks to 77 yr), with the highest incidence during the second decade. Of 12 patients with signs or symptoms resulting from complications of a Meckel's diverticulum, four presented with hemochezia varying from passage of bright red blood to passage of tarry feces. Gastric mucosa was present in the diverticulum in three of these patients, and ileal mucosa was found in the fourth. Abdominal pain was the primary symptom in four other patients, each of whom was found to have acute Meckel's diverticulitis, with or without perforation. The remaining four had small bowel obstruction secondary to either intussusception or an adhesive band. Of the 33 diverticula, 31 were removed at operation. There were no operative deaths. The major complications reported in the literature for Meckel's diverticulum are hemorrhage, obstruction, inflammation, and benign and malignant neoplasia. Hemorrhage is most frequent in children, while inflammation is most common in adults. Because complications of a Meckel's diverticulum occur in 15-33% of cases and invariably require operative treatment, and because when signs or symptoms arise, morbidity and mortality are high, it is suggested that incidental Meckel's diverticulectomy be performed at any age when the morbidity and mortality of the primary procedure will not thus be substantially increased.

5861 ANTIPERISTALTIC ILEAL SEGMENT IN THE PREVENTION OF ILEOSTOMY DIARRHEA. *(E.)*
Cohen, S. E.; Matolo, N. M.; Michas, C. A.; Wolfman, Jr., E. F. (Univ. California Sch. Med., Davis). *Arch. Surg.* 110(7):829-832, 1975.

An antiperistaltic segment was created in 5 of 10 ileostomized dogs to investigate its value in prevention of ileostomy diarrhea. All 10 had the terminal part of the ileum divided about 5 cm from the ileocecal valve, the distal end oversown, and the proximal end exteriorized. In 5 dogs a 4-cm antiperistaltic ileal segment was placed 30.5 cm proximal to the ileostomy. The antiperistaltic segments were removed after 3 weeks, and the intestinal continuity restored. Daily preoperative (5 days) and postoperative determinations were made of body weight and serum and urine electrolyte values (sodium and potassium). Ileostomy drainage was also calculated. Following surgery, the dogs given the ileostomy only lost about 20% of their body weight, and serum sodium decreased by 12-30 mEq/liter. Potassium remained normal. All 5 died between 2-9 days postoperatively of massive water and electrolyte imbalance. The dogs given ileostomy and an antiperistaltic segment lost less than 10% of their body weight, and serum sodium remained normal. The stools became nonwatery, and roentgenography showed greatly prolonged transit time (2 hr 45 min) in contrast to the controls (50 min). After removal of the antiperistaltic segment, the dogs lost an additional 10-15% of their body weight, serum sodium level showed a mean decrease of 15 mEq/liter, and transit time shortened to one hr and 15 min. Two of the 5 subsequently died of water and electrolyte imbalance. It is concluded that inclusion of an antiperistaltic segment can successfully control ileostomy diarrhea.

5862 SMALL-BOWEL OBSTRUCTION SECONDARY TO ENDOMETRIOSIS. *(E.)* Martimbeau, P. W.; Pratt, J. H.; Gaffey, T. A. (Mayo Grad. Sch. Med., Univ. Minn., Rochester). *Mayo Clin. Proc.* 50(5):239-243, 1975.

A review of Mayo Clinic records (1950-1970) disclosed 11 cases of intestinal obstruction due to endometriosis of the small bowel. The endometriosis involved the ileum in all 11 cases and was not found proximal to the ileum in any case; it was necessary to resect a portion of the small intestine in all cases. The ileal endometriosis was restricted to the serosa and the muscular coats, and the obstruction was due to fibrosis and kinking of the bowel. Because the symptoms are not acute or specific, the diagnosis is seldom established prior to laparotomy. If pelvic endometriosis is minimal, intestinal resection is adequate; but if the pelvic lesions are extensive, or if multiple sites in the intestines are involved, then more radical resection and bilateral oophorectomy are indicated.

5863 INTRALUMINAL DUODENAL DIVERTICULUM: REPORT OF TWO CASES AND REVIEW OF THE LITERATURE. *(E.)* Fleming, C. R.; Newcomer, A. D.; Stephens, D. H.; Carlson, H. C. (Mayo Grad. Sch. Med., Univ. Minn., Rochester). *Mayo Clin. Proc.* 50(5):244-248, 1975.

Because of the rarity of intraluminal duodenal diverticulum, 2 cases are presented along with a review of the literature on the subject. In both cases described, a roentgenogram of the upper gastrointestinal tract showed an intraluminal duodenal diverticulum in the 2nd portion of the duodenum. As of 1972, there were approximately 33 reported cases of intraluminal duodenal diverticulum; 29 of the 33 patients were adults. Most patients presented with abdominal pain and obstructive symptoms; gastrointestinal bleeding had occurred in 25%. Barium study showed a characteristic radiolucent halo that represented the wall of the diverticulum. It is suggested that surgery be approached with caution because of possible injury to the papilla, and in children, coexisting congenital anomalies may be present; in general, surgical intervention should be reserved for patients who have severe, persisting symptoms.

5864 GALLSTONE ILEUS: REVIEW OF THE LITERATURE AND PRESENTATION OF THIRTY-FOUR CASES. *(E.)* Day, E. A.; Marks, C. (Louisiana State Univ. Sch. Med., New Orleans). *Am. J. Surg.* 129(5):552-558, 1975.

An analysis of 34 patients (31 women and 3 men, aged 37-93 yr) with gallstone ileus is presented with a review of the literature on the subject. A history of biliary disease was present in 13 of the 34 patients. Surgical treatment was carried out in 32 patients, with an operative mortality of 19%; enterotomy alone had a mortality of 14%. Only 3 of 32 patients who underwent operation had recurrent symptoms of biliary disease. Two patients in the series of 34 developed carcinoma of the gallbladder. All 3 patients who underwent one-stage cholecystectomy, fistula repair, and enterotomy survived. Those 3 cases added to 23 previously reported offer validity to one-stage repair in dealing with the entire disease process during one operative procedure, thus eliminating the high morbidity after enterotomy alone.

5865 LUMENAL AND MEMBRANE HYDROLYSIS OF STARCH IN SOME DISEASES OF THE SMALL INTESTINE AND PANCREAS. *(E.)* Masevitch, C. H.; Ugolev, A. M.; Zabelinskii, E. K.; Kisily, N. P. (Leningrad Med. Inst. Hyg. Sanit., USSR). *Am. J. Gastroenterol.* 63(4):299-306, 1975.

α-Amylase and γ-amylase levels were determined in a study of lumenal and membrane hydrolysis of starch. A total of 143 subjects were studied: 28 normal; 24 with nontropical sprue; 81 with chronic proximal enteritis; 10 with chronic pancreatitis. Mucosal samples of small intestine were aspirated and prepared for both morphological and enzymatic studies. A dependence was observed between the characteristics of the membrane hydrolysis of starch and the degree of morphological disturbances of the intestinal mucosa. The maximal decrease of the binding properties of the intestinal epithelium and γ-amylase of homogenate was found in subtotal atrophy of mucosa. In chronic jejunitis without atrophy the degree of disturbance of the membrane hydrolysis was minimal. An intermediate degree of disturbance was observed in partial atrophy of mucosa. In patients with chronic pancreatitis, α-amylase activity in the lumen of jejunum decreased 75-80%. The characteristics of the membrane digestion of starch were correlated with the morphological pattern of mucosa; the degree of lumenal hydrolysis of starch was approximately normal, with the exception of a decrease in 6 patients. The results suggest that the lumenal and membrane hydrolysis form an integrated system within the limits of which mutual compensation of deficiencies is possible.

5866 VITAMIN B_{12} ABSORPTION IN PATIENTS WITH CONTINENT ILEOSTOMY. *(E.)* Jagenburg, R.; Kock, N. G.; Philipson, B. (Sahlgrenska Hosp., Goteborg, Sweden). *Scand. J. Gastroenterol.* 16(1):141-144, 1975.

Vitamin B_{12} absorption was studied in 16 men and 18 women 10 months to 6 yr after construction of a continent ileostomy. The mean age of the patients at the time of study was 33 yr, and none had been treated with vitamin B_{12}, folate, or iron after the operation. Schilling test values, serum vitamin B_{12}, serum folate, serum iron, total iron binding, capacity (TIBC) and hemoglobin were measured in all patients; in 3 patients, the local absorption of vitamin B_{12} from the ileal reservoir was measured. Normal Schilling test values were obtained in 21 patients after a single determination, and repeated tests showed a normal B_{12} absorption in 8 of the 13 patients in whom the Schilling test values were initially below 10%. In the remaining 5 patients, repeated Schilling tests showed borderline values (5-10%) in 4 cases and a low value (mean 4.5%) in 1 case. One of the patients with borderline values had previously undergone resection of 80 cm of the terminal ileum. In all but 1 patient, the serum iron, TIBC, serum folate, and whole blood folate levels were normal. When radioactive vitamin B_{12} and intrinsic factor were instilled directly into the ileal reservoir, the recovery of radioactivity in the 24-hr urine samples varied from 1.6-6.9%, indicating that the vitamin was absorbed by the mucous membrane of the reservoir. The construction of a continent ileostomy reservoir does not, therefore, appear to expose the patient to an increased risk of developing vitamin B_{12} malabsorption.

5867 MUCOSAL MORPHOLOGY, BACTERIOLOGY, AND ABSORPTION IN INTRA-ABDOMINAL ILEOSTOMY RESERVOIR. *(E.)* Philipson, B.; Brandberg, A.; Jagenbrug, R.; Kock, N. G.; Lager, I.; Ahren, C. (Sahlgrenska Hosp., Goteborg, Sweden). *Scand. J. Gastroenterol.* 16(1):145-153, 1975.

The mucosal morphology, microbial flora, and absorption from continent ileostomy reservoirs were studied in 6 patients provided with such reservoirs after undergoing proctocolectomy because of ulcerative colitis. The studies were performed 1-12 months and 2 yr after construction of the reservoir. Biopsies of the reservoir mucosa were examined by light and electron microscopy; serial dilutions of the reservoir contents were incubated with appropriate media under aerobic and anaerobic conditions; and the absorption of directly instilled test solution (containing L-phenyl-alanine, D-xylose, and phenolsulfonaphthalein) from the reservoir was measured. The results indicated that the mucous membrane of the terminal ileal segment used for construction of the reservoir underwent morphological changes characterized by an increased cell turnover and shortening of the villi; these changes were noted within 1 month after the operation, and there was no evidence for a continuously progressing process. The brush border was intact in all specimens and no cellular lesions were discovered. The number of microorganisms/g of reservoir contents varied between 10^4 and 10^{8-9}. In 19 of the 26 samples the anaerobes outnumbered the aerobes, and in 5 specimens, equal numbers of aerobes and anaerobes were found. *Escherichia coli* and *Streptococcus faecalis* were isolated from all samples, and lactobacilli,

bacteroides, and *Clostridium perfringens* were isolated at least once from 5 of the 6 patients. In spite of the new microbial ecology and morphological changes, the reservoir mucous membrane did not retain an absorptive capacity for D-xylose and phenylalanine.

5868 TRUE VILLOUS ADENOMA OF THE JEJUNUM. *(E.)* Delevett, A. F.; Cuello, R. (Bridgeport Hosp., Conn.). *Gastroenterology* 69(1):217-219, 1975.

A 73-yr-old female presented with intermittent abdominal pain, anorexia, weight loss, and a positive stool hematest. An intraluminal defect in the middle portion of the ascending colon was noted. At operation, a fungating tumor in the ascending colon was found. A portion of terminal ileum, the cecum, and ascending colon was resected and an ileotransverse colostomy was performed. Pathological study showed an adenocarcinoma of the colon with negative regional nodes. Fifteen months later the patient experienced five attacks of constant midabdominal pain and vomited large quantities of greenish fluid which relieved the attacks. Examination revealed intussusception of the jejunum and a tumor mass at the distal end of the intussuscepting bowel. Operation revealed no evidence of recurrent tumor. On opening the jejunum, a soft, thickened, markedly hypertrophic mucosa with definite demarcation from the normal bowel was noted. Part of the jejunum was resected. Studies showed the tumor to be a hyperplastic proliferating adenomatous lesion with a villous pattern, consisting of tall columnar epithelium suspended in long cords of loosely arranged fibrous connective tissue. There was no evidence of atypia or of infiltration of the underlying stroma. This patient demonstrated the typical gross and microscopic characteristics of a villous adenoma.

5869 SMALL INTESTINAL OBSTRUCTION WITH GENERALIZED DIVERTICULA OF THE GASTROINTESTINAL TRACT. *(Ger.)* Brunner, H.; Penner, E.; Kokoschke, R.; Eggerth, G.; Korn, P. (Dep. Gastroenterol. Med. Comput. Sci., Univ. Vienna, Austria). *Wien. Med. Wochenschr.* 125(18):290-292, 1975.

5870 RARE INTESTINAL TUMORS IN CHILDREN. *(Fr.)* Gout, J. P.; Lebranchu, Y.; Rossignol, A. M. (no affil.). *Med. Infant.* 81(5):511-524, 1974.

5871 MECKEL'S DIVERTICULUM IN CHILDREN. *(Fr.)* Rambaud, P.; Varloteaux, C. H.; Bessard, G. (no affil.). *Med. Infant.* 81(5):445-451, 1974.

5872 INTRALUMINAL DIVERTICULUM IN THE DUODENUM. *(Ger.)* Brijs, V. A. (St. Elisabeth Hosp., Heretals, Belgium). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 122(2):171, 1975.

5873 GALLSTONE ILEUS. *(Rus.)* Izmailov, G. A.; Skvortsov, M. G.; Fattakhov, K. S. (Kazan Med. Inst., USSR). *Klin. Med.* 53(2):123-124, 1975.

5874 PECULIARITIES OF THE COURSE OF CHRONIC DUODENITIS IN CHILDHOOD. *(Rus.)* Apostolov, B. G.; Sokolova, M. I.; Paikov, V. L. (Dept. Infantile Dis., Leningrad Sanit.-Hyg. Med. Inst., USSR). *Vopr. Okhr. Materin. Det.* 20(3):19-23, 1975.

5875 PEPTIC ULCER AND GASTRODUODENAL DIVERTICULA. *(Rus.)* Leschenko, V. M.; Alekseenko, A. A. (Fac. Pediatr., Donetsk Med. Inst., USSR). *Sov. Med.* (2):69-73, 1975.

5876 LEFT PARADUODENAL HERNIA. REPORT OF TWO CASES. *(Fr.)* Marchand, M.; Lenriot, J. P.; Hovasse, P. (Cochin Hosp., Paris, France). *J. Chir. (Paris)* 108(5):435-440, 1974.

5877 STUDIES ON THE ACIDITY OF THE GASTROINTESTINAL CONTENT IN ILEUS. *(Rus.)* Cvetanski, I. (Dept. Surg., Fac. Med., Sofia, Bulgaria). *Khirurgiia (Sofiia)* 27(2):117-122, 1974.

5878 COMPLICATIONS IN PRIMARY TUMORS OF THE SMALL INTESTINE. *(Ger.)* Geroulanos, S.; Hahnloser, P. (Canton Hosp., Zurich, Switzerland). *Schweiz. Med. Wochenschr.* 105(18):582-584, 1975.

5879 PEUTZ-JEGHERS SYNDROME. COMPLICATIONS AND SURGICAL THERAPY. *(Ger.)* Fabian, W.; Becker, H. (Northwest. Hosp., Frankfurt/Main-Praunheim, Germany). *Fortschr. Med.* 93(1):5,8, 1975.

5880 COMPLICATIONS OF INTRAMURAL HEMATOMA OF THE DUODENUM. *(Fr.)* Dbjay, H. C.; Letoumeur, F.; Bahuaud, J. (Cent. Hosp., Montauban, France). *Ann. Chir.* 28(12):1031-1040, 1974.

5881 MECHANICAL INTESTINAL OBSTRUCTION. *(Ger.)* Schriefers, K. H. (Kemperhof Munic. Hosp., Koblenz, Germany). *Chirurg* 46(2):49-53, 1975.

5882 A SEDATIVE FOR PAIN IN THE UPPER RIGHT QUADRANT OF THE ABDOMEN AND DUODENAL DYSKINESIA (THIAPRIDE). *(Fr.)* Albot, G. (Hotel Dieu, Paris, France). *Ann. Gastroenterol. Hepatol.* 11(1): 69-72, 1975.

5883 ROENTGENOLOGIC FINDINGS OF PRIMARY RETICULUM CELL SARCOMA. *(Jpn.)* Uchida, H.; Ikeda, H.; Kuroda, C.; Sato, M.; Nakamura, H.; Kozuka, T.; Ishida, O.; Maekawa, T.; Mori, S. (Osaka University Med. Sch., Japan). *Hinsho Hoshasen* 20(2): 137-144, 1975.

5884 INFUSION THERAPY IN INTESTINAL OBSTRUCTION. *(Ger.)* Wenzel, M.; Dohrmann, R. (Behring Munic. Hosp., Berlin, Germany). *Chirurg* 46(2):62-65, 1975.

5885 PARALYTIC INTESTINAL OBSTRUCTION. *(Ger.)* Rossetti, M. (Dep. Surg., Univ. Basel, Switzerland). *Chirurg* 46(2):54-56, 1975.

5886 GIGANTIC ULCERATED DIVERTICULUM IN THE THIRD PART OF THE DUODENUM. *(Sp.)* Lopez Gallart, J. M. (Gallart-Mones Sch. Dig. Pathol., Barcelona, Spain). *Rev. Esp. Enferm. Appar. Dig.* 45(4):417-424, 1975.

5887 A CASE OF TUBERCULOSIS OF THE SMALL INTESTINE. *(Jpn.)* Shida, T.; Maeda, Y.; Kuroda, Y.; Motai, Y.; Miyoshi, A.; Morimatsu, M. (Chihaya Hosp., Fukuoka City, Japan). *Rinsho Hoshasen* 20(1):51-56, 1975.

5888 RADIOLOGICAL FINDINGS OF THE SMALL INTESTINE IN THE SHÖNLEIN-HENOCH SYNDROME. *(Jpn.)* Yamazaki, M.; Sato, K.; Yamamoto, M.; Koike, T. (Niigata Univ. Med. Sch., Japan). *Rinsho Hoshasen* 20(2):145-152, 1975.

5889 CONSERVATIVE THERAPY OF ILEAL OBSTRUCTION: ENDOSCOPIC AIDS IN TREATMENT. *(Ger.)* Doehn, M.; Rehner, M.; Soehendra, N.; Wehling, H. (Surg. Clin. Polyclin., Univ. Hamburg, Germany). *Dtsch. Med. Wochenschr.* 100(22):1249-1250, 1975.

5890 DESCRIPTION OF 2 CASES OF TRAUMATIC DUODENAL RUPTURE. *(Fr.)* Nguyen-Van-Hanh. (Viet Duc Hosp., Vietnam). *Radiol. Diagn. (Berl.)* 15(4):615-617, 1974.

5891 DUODENAL OBSTRUCTION RELATED TO BENIGN BILIARY TRACT DISEASE. *(E.)* Grace, D. M.; Himal, H. S. (R. Victoria Hosp., Montreal, Canada). *Can. J. Surg.* 18(4):345-348, 1975.

5892 BACTERIAL PERITONITIS AND THE BURSTING STRENGTH OF INTESTINAL ANASTOMOSES. *(E.)* Kilam, S. K.; Jackson, F. L.; Williams, H. T. G. (Dep. Surg., Mem. Univ. Newfoundland, St. Johns). *Can. J. Surg.* 18(4):372-378, 1975.

5893 TREATMENT OF CHOLERHEIC DIARRHEA WITH CHOLESTYRAMINE AND A HYDROPHILIC COLLOID. *(E.)* Thompson, W. G. (Ottawa Civic Hosp., Canada). *Dis. Colon Rectum* 18(4):304-307, 1975.

5894 FACTORS DETERMINING WEIGHT LOSS AFTER JEJUNOILEAL BYPASS FOR OBESITY. *(E.)* Guzman, I. J.; Varco, R. L.; Buchwald, H. (Univ. Minnesota Hosp., Minneapolis). *J. Surg. Res.* 18(4): 399-403, 1975.

5895 PNEUMATOSIS CYSTOIDES INTESTINALIS WITH UNEXPLAINED ASCITES IN ETHIOPIAN PATIENTS. *(E.)* Tsega, E. (St. Paul's Hosp., Addia Ababa, Ethiopia). *Br. J. Surg.* 62(5):379-382, 1975.

5896 JEJUNAL-ILEAL SHUNT IN THE TREATMENT OF MORBID OBESITY. *(E.)* Richards, V. (Child Hosp., San Francisco, Calif.). *Bull. Soc. Int. Chir.* 33(5-6):364-370, 1975.

5897 THE ROLE OF FIBEROPTIC ENDOSCOPY IN THE DIAGNOSIS AND MANAGEMENT OF DUODENAL NEOPLASMS. *(E.)* Wald, A.; Milligan, F. D. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *Am. J. Dig. Dis.* 20(6):499-505, 1975.

5898 REVERSED JEJUNAL SEGMENT USED TO TREAT THE SHORT BOWEL SYNDROME. *(E.)* Hakami, M.; Moslehy, A.; Mosavy, S. H. (Isfahan Univ. Sch. Med., Iran). *Am. Surg.* 41(7):432-435, 1975.

5899 METABOLIC STUDIES BEFORE AND AFTER JEJUNOILEAL BYPASS FOR MASSIVE OBESITY. *(E.)* Weismann, R. E. (Dartmouth Med. Sch., Hanover, N.H.). *Bull. Soc. Int. Chir.* 33(5-6):371-377, 1974.

5900 FURTHER CONSIDERATIONS IN USE OF JEJUNOILEAL BYPASS IN PATIENTS WITH MORBID OBESITY. *(E.)* Scott, Jr., H. W.; Dean, R. H. Shull, H. J.; Gluck, F. W.; Abram, H. S.; Webb, W.; Younger, R. K.; Brill, A. B. (Vanderbilt Univ. Med. Cent., Nashville, Tenn.). *Bull. Soc. Int. Chir.* 33(5-6):378-387, 1974.

See also, 5587, 5588, 5635, 5652, 5655, 5656, 5660, 5667, 5684, 5686, 5687, 5711, 5913, 5917, 5921, 5938, 6022, 6056, 6071, 6204, 6390, 6395, 6403, 6418, 6476, 6484, 6505, 6519, 6520, 6536, 6542, 6591, 6592.

SMALL INTESTINE

Malabsorption

5901 LEUKOCYTE REACTIVITY TO α-GLIADIN IN DERMATITIS HERPETIFORMIS AND ADULT COELIAC DISEASE. *(E.)* Allardyce, R. A.; Shearman, D. J. C. (Univ. Dep. Ther., Edinburgh, Scotland). *Int. Arch. Allergy Appl. Immunol.* 48(3):395-400, 1975.

The leukocyte adherence inhibition test was used to study leukocyte reactivity to α-gliadin in 20 patients with dermatitis herpetiformis, and in 11 patients with adult celiac disease. Sedimented peripheral blood leukocytes from patients and normal

subjects were washed twice in Hepes-buffered essential medium containing Hank's salts, and incubated in plastic tubes with 100 µg of purified α-gliadin at a concentration of 2 x 10^7 cells/ml. Control preparations were incubated without antigen. Following incubation for 30 min at 37 C, the test and control cells from each individual were placed on opposite sides of duplicate non-mirrored surfaces. After further incubation for 1 hr at 37 C in a moist atmosphere, the cells were counted. The coverslips were removed, and the non-adherent cells were removed. After a drop of fresh medium was placed on the surface, the remaining adherent cells were counted. The percent adherence of cells from the controls and test slides of each surface was calculated, and the mean of duplicate readings was recorded. The percent inhibition of leukocyte adherence equals 100 times the mean test adherence over the mean control adherence. Leukocytes from both groups of patients showed a significant inhibition of leukocyte adherence. Possible cell-mediated reactivity to this fraction of gluten is suggested.

5902 THE UPTAKE OF VITAMIN B_{12} BY THE SEDIMENT OF JEJUNAL CONTENTS IN PATIENTS WITH THE BLIND-LOOP SYNDROME. *(E.)* Schjonsby, H.; Hofstad, T. (Med. Dep. A, Gade Inst., Univ. Bergen, Norway). *Scand. J. Gastroenterol.* 10(3):305-309, 1975.

Whether the sediment of jejunal contents in patients with the blind-loop syndrome was able to take up intrinsic factor-bound vitamin B_{12}, and whether such uptake prevented the intestinal absorption of the vitamin, was studied. Following preincubation of intrinsic factor-bound $^{57}CoB_{12}$ with the jejunal sediments of six patients with the blind-loop syndrome, the mean uptake by the sediments of intrinsic factor-bound $^{57}CoB_{12}$ (28.1%) was significantly higher than the mean uptake by jejunal sediments from five control patients (5.8%). The uptake by the sediments significantly decreased when the incubations were carried out in the presence of lincomycin and neomycin. The jejunal sediments from the patients with the blind-loop syndrome inhibited the uptake of intrinsic factor-bound $^{57}CoB_{12}$ by perfused rat intestinal segments, whereas the sediments from controls had no such inhibitory effects. The results indicate that uptake by intestinal bacteria of intrinsic factor-bound vitamin B_{12} may contribute to the malabsorption of the vitamin in patients with the blind-loop syndrome, since the B_{12}-intrinsic factor complex is unavailable for intestinal absorption when it has been taken up by intestinal bacteria.

5903 MALABSORPTION AND PROTEIN-LOSING ENTEROPATHY IN A CHILD WITH X-LINKED AGAMMAGLOBULINEMIA. *(E.)* Norman, M. E.; Hansell, J. R.; Holtzapple, P. G.; Parks, J. S.; Waldmann, T. A. (Child. Hosp. Philadelphia, Pennsylvania). *Clin. Immunol. Immunopathol.* 4(2):157-164, 1975.

A patient with infantile x-linked agammaglobulinemia was studied for malabsorption and protein-losing enteropathy. He first presented for hospitalization at 3 yr of age with severe hypogammaglobulinemia, a nonbloody diarrhea, vomiting, and arthralgia. Otitis media and sinusitis were present and treated with ampicillin. The patient showed failure to thrive over the next 30 months with intermittent diarrhea and anorexia. At 6 yr of age, diarrhea and weight loss became more severe despite gamma globulin therapy, but plasma therapy caused a temporary remission. At 7 1/2 yr, hypocalcemic tetany developed and malabsorption secondary to gluten enteropathy was diagnosed. Gluten-free diet caused a temporary remission. At 8 1/2 yr, radiography revealed thickening of the mucosal folds of the upper small bowel, and segmentation of the barium. Biopsy showed slightly flattened villi and inflammatory cells in the lamina propria. Functional hypoparathyroidism secondary to hypomagnesemia was later diagnosed, and magnesium therapy controlled the tetany. At 10 yr, an episode of severe hypoprothrombinemia occurred and the malabsorption syndrome was reevaluated. For protein metabolic studies, ^{51}Cr-albumin and ^{125}I-IgG were given i.v. at 14 and 13 µCi, resp., and 24 hr urine and stool collections were made from days 1-7. Immunologic studies involved measurement of immunoglobulins G, M, A and E and of CH50 hemolytic and C_3 complements. Delayed hypersensitivity skin tests were performed and an *in vitro* assay of cellular immunity was carried out. The labelled IgG had a half-life of 9.0 days and showed a fractional catabolic rate of 17.9%. Actual plasma loss was about 58.8 ml/day based on the ^{51}Cr-albumin, significantly higher than normal. Immunoglobulin levels were markedly reduced (IgG: 48-100, IgA: 0-5, IgM: 0-9 mg/100 ml, and IgE: 4 ng/ml); C_3 complement was slightly reduced (86 mg/100 ml). Delayed hypersensitivity testing showed normal response to *Candida* antigen, implying intact cellular immunity. It is concluded that malabsorption and protein-losing enteropathy were both present in a child with infantile x-linked agammaglobulinemia, leading to marked abnormality in gamma globulin metabolism.

5904 PROTEIN-LOSING ENTEROPATHY AND MALABSORPTION IN ACUTE MEASLES ENTERITIS. *(E.)* Dossetor, J. F. B.; Whittle, H. C. (Ahmadu Bello Univ. Hosp., Zaria, Nigeria). *Br. Med. J.* 2:592-593, 1975.

The association of measles and malnutrition was studied in 28 children with acute measles accompanied by diarrhea. Fecal protein loss, urine protein, and lactose tolerance were determined, and 1-hr blood xylose tests were performed. ^{59}Fe-labeled iron dextran (0.1 µCi/kg) was injected i.v.; all stools were collected for three days and plasma was sampled daily. Mean fecal ^{59}Fe-dextran clearance of 5 patients in the acute stage was 5.7% of plasma volume/day. After recovery, clearance fell to 1.49%. Absolute albumin loss was 1.68 g/day. Mean urine protein loss in 22 patients in the acute stage was 350 mg/liter, most of which was albumin. None showed urinary protein on recovery. Of 17 patients tested for lactose intolerance, 4 were intolerant. One died; the others were tolerant on retesting. Blood glucose rose more than 1.67 mM/liter in 11 and rose 1.11-1.67 mM/liter in 5. Two of the latter died.

In 1 patient, blood glucose did not rise during illness but rose 1.17 mM/liter on recovery. Xylose absorption tests showed a mean acute level of 0.93 mM/liter and a recovery level of 1.71. It is concluded that fecal protein loss and malabsorption may contribute significantly to malnutrition following measles.

5905 DERMATITIS HERPETIFORMIS AND SMALL INTESTINAL LESION - NO STRICT ASSOCIATION IN GERMAN PATIENTS. *(E.)* Ruppin, H.; Weidner, F.; Domschke, S.; Domschke, W.; Classen, M.; Hornstein, O. P. (Dep. Med., Univ. Erlangen-Nurnberg, Germany). *Acta Hepatogastroenterol.* 22(2):105-111, 1975.

An investigation was conducted of morphological and functional characteristics of small intestinal mucosa in a group of 16 German patients with dermatitis herpetiformis (8 men and 8 women, aged 41-79 yr) and in healthy German controls living in the same Bavarian area of Erlangen-Nurnberg and its immediate vicinity. By stereomicroscopy, finger- or leaf-shaped villi were found exclusively both in patients and controls. Convoluted or flat mucosa could not be detected in dermatitis herpetiformis patients. Accordingly, in all cases, normal structure of jejunal mucosa without signs of mucosal or submucosal inflammation was demonstrated histologically. However, mean counts of interepithelial lymphocytes in dermatitis herpetiformis patients were slightly but significantly increased over controls. Intestinal disaccharidase activities were not statistically different from those of healthy control subjects, and oral lactose tolerance tests were normal in each of 8 patients tested. It is suggested that immunological and genetic differences between dermatitis herpetiformis patients reported in British and American papers and the patients studied in this report may be responsible for the basically differing results as to "dermatogenic enteropathy". Moreover, the mean ages of patients reported in other studies were about 19 to 26 yr below the mean age found in this study. It is suggested that the age of first manifestation and follow-up of dermatitis herpetiformis may also be important factors in the nature and frequency of intestinal involvement in dermatitis herpetiformis.

5906 PREGNANCY IN PATIENTS WITH COELIAC DISEASE. *(E.)* Ogborn, A. D. R. (Hammersmith Hosp., London, England). *Br. J. Obstet. Gynaecol.* 82(4):293-296, 1975.

The effects of dietary management and supportive obstetric care on the course and outcome of pregnancies in patients with celiac disease were studied. Twenty-five women, who had previously gone through a total of 38 pregnancies, were placed on a strict gluten-free diet and given calcium gluconate twice daily (1.5 g, p.o.), vitamin B_{12} (1000 µg, i.m.) after 6, 20, and 34 weeks of gestation, and compound i.m. vitamins once weekly. Exacerbations of diarrhea were controlled by hospital rest, dietary supervision, and salicylazosulfapyridine or codeine phosphate as necessary. Progesterone status was monitored during the first 16 weeks and hydroxyprogesterone (250 mg twice weekly) given as indicated. Fetoplacental function was monitored during the third trimester and the fetus was closely monitored during labor. The perinatal mortality in patients on an unrestricted diet was 7%, 74% of the 38 pregnancies in women on unrestricted diets ending in the birth of a viable infant which survived the neonatal period. The perinatal mortality rate in the treated patients was 10%, 86% of the pregnancies in this group ending in the birth of a viable infant which survived the neonatal period. None of the babies had celiac disease. Although no significant differences were demonstrable between the treated and untreated groups, the apparent overall improvement in outcome of the pregnancies with dietary management and obstetric supervision was encouraging.

5907 IN VITRO PATHOGENETIC STUDIES OF COELIAC DISEASE: EFFECTS OF PROTEIN DIGESTS ON COELIAC INTESTINAL BIOPSY SPECIMENS MAINTAINED IN CULTURE FOR 48 HOURS. *(E.)* Jos, J.; Lenoir, G.; de Ritis, G.; Rey, J. (Hosp. Enfants-Malades, Paris, France). *Scand. J. Gastroenterol.* 16(1):121-128, 1975.

The effects of various gliadin and casein digests on celiac intestinal biopsy specimens maintained in culture for 48 hr were studied. Biopsy specimens were taken at the duodeno-jejunal flexure from 11 control children, 24 children with treated celiac disease, and 20 children with untreated celiac disease. The specimens were incubated at 37 C for 48 hr in media containing antibiotics and, in some cases, various peptic-tryptic (PT) or peptic-tryptic-chymotryptic (PTC) digests of gliadin and casein. The specimens were examined immediately after excision and after 48 hr incubation by light and electron microscopy. The morphologic abnormalities in the surface epithelial cells in 11 of the 14 biopsy specimens from children with active celiac disease disappeared almost immediately during culture in the absence of gliadin digests. In flat biopsies cultured in the presence of the PT gliadin digest, epithelial restoration was inhibited and a cytotoxic effect was obvious; a similar effect was observed in the presence of the PTC gliadin digest and the autoclaved PT gliadin digest. The casein hydrolysates exerted a slight toxic effect which seemed to be reduced by autoclaving. In the control specimens and in 16 restored celiac biopsies from patients in remission, there was no or little change after culture in the presence of gliadin or casein digests. In 6 of 11 biopsy specimens taken from 8 patients in remission after gluten challenge, PT, autoclaved PT, and PTC gliadin digests all exerted an obvious cytotoxic effect; in most cases, no such effect was seen in the presence of the PT digest of casein. In all specimens studied, thorough PTC hydrolysis of gliadin abolished all of its *in vitro* toxic effects. The results suggest that the toxicity of gliadin is related to a relatively large peptide, which supports the hypothesis of a basic immune process.

5908 MACROAMYLASEMIA ASSOCIATED WITH MALABSORPTION AND CRYOGLOBULINEMIA: CLINICAL AND BIOLOGICAL STUDY OF A NEW CASE. *(Fr.)* Touboul, J. P.; Hadchouel, P.; Hirsch-Marie, H.; Caroli, J.; Renault, H. (St. Antoine Hosp., Paris, France). *Med. Chir. Dig.* 3(6):419-426, 1974.

A new case of macroamylasemia associated with malabsorption and cryoglobulinemia was reported in a 33-yr-old woman with persistent and marked elevated amylasemia contrasted by normal amylasuria. The patient also suffered from severe weight loss (15 kg), asthenia, diarrhea and Raynaud's phenomenon. The macroamylase, demonstrated by serum electrophoresis and chromatography, was an amylase-IgA complex. Malabsorption, verified by various tests (quantitative analysis of fat in feces, induced hyperglycemia, lactose test, D-xylose test), may explain the anomalies observed in the hepatic picture (lowered cholesterol and lowered coagulation factors). Cryoglobulinemia, which was not involved in macroamylasemia, was due to the presence of IgA. Hematological results showed marked lymphoplasmocytic-type medullary infiltration. Although the patient did not exhibit classical dysglobulinemia, the coincidence of malabsorption and lymphoplasmocytic-type medullary infiltration might lead to the eventual appearance of a system disease. A review of the literature seems to indicate the relatively frequent association between IgA-macroamylasemia, malabsorption, alcoholism and pancreatitis.

5909 FOLLOW-UP STUDY OF PATIENTS WITH CELIAC DISEASE. *(Ger.)* Emons, D.; Bohm, E. R.; Rotthauwe, H. W. (Pediatr. Clin., Univ. Bonn, Germany). *Dtsch. Med. Wochenschr.* 99(38):1847-1853, 1974.

Follow-up examinations were performed on 25 children (ten boys and 15 girls) who had been given diet therapy for celiac disease diagnosed clinically before 3 yr of age on the basis of retarded development, distended abdomen, and pathologically loose stools (recurrent diarrhea in almost all cases, but abnormally bulky stools in only a few). In 13 cases the diagnosis was considered confirmed on the basis of abnormal xylose tests and prompt response to a gluten-free diet. Anemia, which often occurs in the first few weeks after institution of diet therapy, developed in 21. Life-threatening toxicosis occurred in four patients and was associated with hypoproteinemia and protein-deficiency edema in one. Cystic fibrosis was ruled out in all 25 children by the sweat test. Enteropathogenic bacteria were present initially or intercurrently in eight cases, and radiological examinations revealed the presence of functional gastrointestinal disorders in seven of the eight patients examined. Osteoporosis was radiologically diagnosed in seven children, but none had radiological or biochemical evidence of manifest rickets. At follow-up, villous atrophy was found on duodenal biopsies in 20 of these 25 children, and another patient developed marked villous atrophy 9 months after diet therapy was discontinued. The intestinal mucosa was completely normal in four cases. Patients with villous atrophy had eaten gluten-free diets for 6 months to 5 yr (mean 2 yr), and had eaten normal food for 9 months to 8 yr before biopsy. At follow-up, only one of these 21 patients had clinical and biochemical signs of a recurrence; although a long-term, gluten-free diet was prescribed, the parents failed to cooperate. Only three of these 21 children had to be treated for intestinal disorders when they started eating normal food. Clinical findings, hemoglobin levels, stool fat contents, and xylose test were normal on all but one of the 21 children with persistent villous atrophy at follow-up.

5910 TOTAL BODY POTASSIUM IN CHILDREN, WITH PARTICULAR REFERENCE TO COELIAC DISEASE. *(E.)* Boddy, K.; King, P. C.; Carswell, F. (Scottish Univ. Res. Reactor Cent., Glasgow). *Clin. Sci. Mol. Med.* 49(2):133-137, 1975.

Total body potassium was measured by whole-body counting in 20 children (11 males and nine females, 8 months to 13 yr and 10 months of age) with celiac disease and was compared with similar measurements in 20 children having diseases in which there was no clinical suspicion of electrolyte or water disturbance. The controls were matched as closely as possible with respect to sex, age, and height. All children were on a gluten-containing diet at the time of measurement. Total body potassium was estimated by detecting the 1.46 MeV gamma rays of ^{40}K, which is naturally radioactive and a constant fraction of natural body potassium. There was no significant difference between the measured values of total body potassium (in mM) in children with celiac disease and in control children or between males and females. At the clinical level, infantile celiac disease does not parallel malnutrition with respect to body potassium; potassium deficiency is not a complication of this disease. It is suggested that the potassium deficiency in malnutrition is a direct result of dietary factors. Apparently, in celiac disease, the potassium presented for absorption is evidently handled normally or there is compensatory retention by the body to accomodate abnormalities in absorption, giving a long-term normal balance.

5911 A CASE INDICATIVE OF THE EXISTENCE OF A CONNATAL FORM OF WHIPPLE'S DISEASE. *(It.)* De Pra, M.; Casagrande, A.; Guarino, M. (Dept. Pediatr., Tirano Gen. Hosp., Italy). *Minerva Pediatr.* 26(34): 1723-1743, 1974.

5912 DIAGNOSIS OF CELIAC DISEASE IN A SERIES COVERING THE YEARS 1969-1973. *(It.)* Ansaldi, N.; Santini, B.; Barbera, C.; Bianchini, O.; Oderda, G. (Pediatr. Clin. II, Univ. Turin, Italy). *Minerva Pediatr.* 26(35):1757-1764, 1974.

5913 REGENERATION OF THE MUCOUS MEMBRANE OF THE SMALL INTESTINE IN SELECTED CASES OF MALABSORPTION SYNDROME. *(Pol.)* Orlowska, J.; Ratajczak-Majewska, C. (CMPK, Warsaw, Poland). *Patol. Pol.* 25(4):603-610, 1974.

5914 MALABSORPTION SYNDROME WITH ACQUIRED AGAMMAGLOBULINEMIA AND SELECTIVE IgA DEFICIENCY WITH CIRCULATING B-LYMPHOCYTES. *(Ger.)* Knolle, J.; Bolte, J. P.; Hopf, U.; Olbermann, M.; Meyer zum Buschenfelde, K. H. (Med. Clin. II, Univ. Mainz, Germany). *Z. Gastroenterol.* 13(3):387-392, 1975.

5915 WHIPPLE'S DISEASE. GASTROINTESTINAL BIOPSY FINDINGS AND HISTOCHEMISTRY IN ONE CASE. *(It.)* Cheli, R.; Ramasso, M.; Nicolo, G.; Aste, H. (Munic. Hosp., Genoa, Italy). *Minerva Gastroenterol.* 20(2):77-78, 1974.

5916 CLINICAL EXPERIENCE WITH HYLAKOMBUN (5,7-DICHLORO-8-HYDROXYQUINOLIN) IN GASTROENTEROLOGY. *(Ger.)* Kocian, V. J.; Kocianova, J. (Inst. Med. Res., Prague, Czechoslovakia). *Wien. Med. Wochenschr.* (Suppl. 25):1-8, 1975.

5917 INTESTINAL MALABSORPTION DUE TO MULTIPLE DIVERTICULOSIS OF THE DUODENUM AND SMALL INTESTINE: REPORT OF A CASE. *(Sp.)* de Aguiar Gonzalez, A. R.; Santillana Lopez, T.; Pardo Redondo, A. (San Carlos Hosp. Clin., Madrid, Spain). *Rev. Clin. Esp.* 134(5):485-490, 1974.

5918 SELECTIVE VITAMIN B_{12} MALABSORPTION (IMMERSLUND-GRASBECK SYNDROME) AS A CAUSE OF DEVELOPMENTAL DISORDERS IN EARLY CHILDHOOD. *(Ger.)* Harms, K.; Bohne, A.; Kollmann, D. (Pediatr. Clin., Univ. Munich, Germany). *Monatsschr. Kinderheilkd.* 123(5):428, 1975.

5919 DIAGNOSIS AND THERAPY OF CONGENITAL AND INHERITED DISACCHARIDASE DEFICIENCIES OF THE SMALL INTESTINAL MUCOUS MEMBRANE. *(Ger.)* Niessen, K. H.; Brugmann, G.; Schmidt, K. (Child. Clin., Univ. Tubingen, Germany). *Monatsschr. Kinderheilkd.* 123(5):429-431, 1975.

5920 CORRELATION IN THE DIAGNOSIS OF INTESTINAL LACTASE DEFICIENCY BETWEEN THE RADIOLOGICAL METHOD AND THE LACTOSE TOLERANCE TEST. *(E.)* Lisker, R.; Lopez Habib, G.; Mora, M. A.; Pitol, A. (Nat. Inst. Nutr., Tlalpan, Mexico). *Rev. Invest. Clin.* 27(1):1-5, 1975.

5921 GLUTEN-SENSITIVE ENTEROPATHY AND INTESTINAL LYMPHORETICULOSIS. A CASE REPORT. *(E.)* Fehmers, M. C. O.; Wilderink, F.; Oushoorn, H. H.; Tytgat, G. N. (Dep. Med., Univ. Amsterdam, Netherlands). *Neth. J. Med.* 18(2):83-88, 1975.

5922 A STUDY OF INTESTINAL ABSORPTION IN SUBJECTS WITH PLURIDISTRICTUAL ATHEROSCLEROSIS. *(E.)* Bracale, G.; Budillon, G.; Squame, G.; Masciariello, S.; Gagliardi, C.; Cimino, L.; Mazzacca, G. (Fac. Med., Univ. Napal, Italy). *Surg. Ital.* 4(1):14-19, 1974.

5923 REVERSIBLE INFERTILITY IN MALE COELIAC PATIENTS. *(E.)* Baker, P. G.; Read, A. E. (Dep. Med., Univ. Bristol, England). *Br. Med. J.* 2(5966):316-317, 1975.

5924 CORRELATION OF LACTASE ACTIVITY, LACTOSE TOLERANCE AND MILK CONSUMPTION IN DIFFERENT AGE GROUPS. *(E.)* Lebenthal, E.; Antonowicz, I.; Shwachman, H. (Harvard Med. Sch., Boston, Mass.). *Am. J. Clin. Nutr.* 28(6):595-600, 1975.

5925 XYLOSE ABSORPTION TEST BEFORE AND AFTER SURGICAL REPAIR OF COARACTATION OF THE AORTA. *(E.)* Markiewicz, A.; Wojczuk, D.; Zmudzinski, J. (Silesian Sch. Med., Katowice, Poland). *Acta Hepatogastroenterol.* 22(2):112-117, 1975.

5926 AN ASSESSMENT OF GASTRO-INTESTINAL AND ENDOCRINE FUNCTIONS. IN PATIENTS WITH THE MALABSORPTION SYNDROME. *(E.)* Tripathi, S. N.; Misra, A. K.; Srivastava, S. K. (Inst. Med. Sci., Banaras Hindu Univ., Varanasi, India). *Am. J. Proctol.* 26(3):79-86, 1975.

See also, 5205, 5670, 5672, 5691, 5866, 6497, 6506, 6548, 6586.

LARGE INTESTINE

5927 SOCIAL CLASS DIFFERENCES AMONG PATIENTS WITH LARGE-BOWEL CANCER IN CALI, COLOMBIA. *(E.)* Haenszel, W.; Correa, P.; Cuello, C. (Natl. Cancer Inst., Bethesda, Md.). *J. Natl. Cancer Inst.* 54(5):1031-1035, 1975.

Social class differences associated with risk of large-intestine cancer were studied based on data from a population-based cancer registry in Cali, Colombia. The analysis covered 214 newly diagnosed cases of adenocarcinoma of the large intestine registered among residents of Cali between 1962-1971. Findings on social class differences in the prevalence of intestinal polyps in 404 autopsies among Cali residents were also included, and measurements were made on fresh colon specimens taken from 4 persons dying from accidental causes to examine the relation between tissue at risk and cancer incidence by segment. The risk of large-intestine cancer in males and females of all ages was higher

for persons in the upper and middle socioeconomic classes than for those of the two lower socioeconomic classes. Risk of cancer of the stomach and uterine cervix was inversely related to social class. The socioeconomic gradients associated with risk of large bowel cancer were most marked for cancer of the ascending and rectosigmoid colon and were minimal for cancer of the cecum and rectum. These data present several parallels with information derived from comparisons of developed and developing countries and also appear consistent with recent information on the possible role of dietary factors in intestinal cancer. It is likely that strong associations between specific dietary items and income facilitated the detection of a socioeconomic gradient for large-intestine cancer in Cali.

5928 GUT BACTERIA AND THEIR METABOLIC ACTIVITIES IN FAMILIAL POLYPOSIS. *(E.)* Bone, E.; Drasar, B. S.; Hill, M. J. (Cent. Public Health Lab., London, England). *Lancet* 1(7916):1117-1120, 1975.

Early work suggested that patients with cancer of the large intestine carry clostridia capable of dehydrogenating the steroid nucleus and also have high fecal bile-acid concentrations. Therefore, colonic environmental factors were investigated in familial polyposis, a disease predisposing to colorectal cancer. Samples of rectal contents and urine were obtained from 6 affected children of polyposis patients, 13 unaffected children of polyposis patients, 27 polyposis patients who had been treated by colectomy and ileorectal anastomosis, 11 ulcerative colitis patients treated by colectomy and ileorectal anastomosis, 8 patients treated for colonic carcinoma by partial colectomy and anastomosis, and 3 patients with benign polyposis syndromes not thought to predispose to malignancy. Although a high proportion of the affected and unaffected children of polyposis patients carried clostridia able to dehydrogenate the steroid nucleus, their average rectal bile-acid concentration was low. In addition, the bile acids were in many cases poorly degraded and were characterized by the presence of chenodeoxycholic acid; in 11 of these patients, including half of the unaffected children, less than 25% of the fecal cholesterol had undergone conversion to coprostanol or coprostanone by the gut flora, although these flora were able to degrade cholesterol *in vitro*. In the treated polyposis patients, rectal bile-acid and neutral-steroid concentrations were low and the cholesterol was virtually undegraded; the cholesterol had undergone some degradation in all of the ulcerative colitis patients who had undergone similar treatment. The results suggest that the inability of the polyposis patients and their unaffected children to degrade cholesterol in the intestine was due to unfavorable conditions in the intestine, a hypothesis that is favored by the lack of cyclic secondary amines in the urine of these persons (these amines are also synthesized by bacteria in the gut). If the unaffected children who were not able to degrade cholesterol later develop polyposis, the degradation of cholesterol by the gut flora would appear to be a simple diagnostic test for persons at high risk of developing polyposis.

5929 PNEUMATOSIS CYSTOIDES INTESTINALIS: TREATMENT WITH OXYGEN VIA CLOSE-FITTING MASK. *(E.)* Simon, N. M.; Nyman, K. E.; Divertie, M. B.; Rovelstad, R. A.; King, J. E. (Mayo Clin., Rochester, Minn.). *JAMA* 231(13):1354-1356, 1975.

Treatment of four patients (2 men and 2 women, aged 64-69 yr) suffering from pneumatosis cystoides intestinalis with oxygen via a close-fitting mask is described. Symptoms included frequent, watery bowel movements, rectal bleeding, flatulence, and abdominal pain. The patients were treated with 70% oxygen administered by close-fitting mask for 5-8 days, after which all symptoms ceased and proctoscopic and barium-contrast roentgenographic findings in the colon were normal. Six months after oxygen treatment, one woman developed slight rectal bleeding which was confirmed as pneumatosis cystoides intestinalis in the rectosigmoid. The patient again responded favorably to oxygen therapy. Although pulmonary damage may result from oxygen exposure, even in patients with normal lungs, prompt withdrawal of oxygen therapy is generally effective in restoring normal pulmonary function within 1-2 days. In the absence of major disturbances of ventilation and perfusion, intermittent administration of 70% oxygen via close-fitting mask should be capable of increasing the nitrogen gradient between a closed space and capillary blood to a degree sufficient to ensure rapid diffusion from one to the other. During daytime meals and at other brief intervals, nasal cannulas may be used to deliver oxygen at 3-4 liters/min. On a program of this kind, with monitoring of vital capacity, the possibility of toxic effects from oxygen is greatly diminished, while the therapeutic needs of the patient can be met effectively. Oxygen treatment can be justified only for patients who have the symptomatic form of pneumatosis cystoides intestinalis and follow-up of patients treated in this manner is indicated.

5930 THE SPECTRUM OF COLITIS ASSOCIATED WITH LINOMYCIN AND CLINDAMYCIN THERAPY. *(E.)* Le Frock, J. L.; Klainer, A. S.; Chen, S.; Gainer, R. B.; Omar, M.; Anderson, W. (West Virginia Univ. Med. Cent. Hosp., Morgantown). *J. Infect. Dis.* 131(Suppl):108-115, 1975.

The clinical, laboratory, sigmodoscopic, x-ray, and biopsy findings in 10 patients (7 women and 3 men, aged 22-70 yr) who developed colitis after lincomycin or clindamycin therapy are reported. Prior to antibiotic therapy in standard dosages, none of the patients had prior histories suggestive of preexisting bowel disease. An abrupt onset of diarrhea, crampy abdominal pain, fever, and leukocytosis was observed 1-12 days after discontinuation of the drug. Proctoscopic examination revealed erythematous friable mucosa covered with small raised, yellowish-white plaques that were sometimes confluent. Barium contrast studies of the colon demonstrated irregular, shaggy mucosa, ulcerations, cobblestone appearances, and thumb printing. Rectal biopsy showed acute inflammation with pseudomembranes with focal or superficial ulcerations. The clinical course lasted an average of 4 weeks, although all 10 patients re-

covered with supportive management consisting of administration of parenteral fluids and electrolytes supplemented with albumin. Antibiotic therapy in such cases is contraindicated unless secondary infection is present, and adrenocorticotropin hormone and steroids appeared to be of little value in modifying the course of the disease. Follow-up barium enemas and proctoscopy were done on all patients after supportive treatment and were normal. A history of diarrhea, fever, and mucosal changes seen on proctoscopy in a patient who has recently received clindamycin or lincomycin should raise the possibility of therapy-associated colitis, and clindamycin should be used only when indicated in serious infections.

5931 THE DETECTION OF ABERRANT DNA SYNTHESIS IN A MEMBER OF A HIGH-RISK CANCER FAMILY. *(E.)* Deschner, E. E.; Long, F. C.; Katz, S. (Mem. Sloan-Kettering Cancer Cent., New York, N.Y.). *Am. J. Dig. Dis.* 20(5):418-424, 1975.

Examination of 4 siblings of a patient with multiple polyposis was performed with radiological, sigmoidoscopic, cytologic, and isotopic labeling procedures for the possible presence of colonic polyps. The patients, 3 men and 1 woman, were all over 40 yr of age. Polyps were absent on films as well as on endoscopy, and colonic cytologies of all 4 subjects were within normal limits. However, isotopic incorporation studies revealed the presence of an abnormal labeling pattern in some crypts of the biopsy incubated with tritiated thymidine of 1 family member. Along with normal crypts with label in the lower 2/3 of the colonic crypts, many crypts had cells labeled at the surface. Some of the surface cells removed by colonic wash were also isotopically labeled; thus, a defect in the regulation of colonic epithelial cell replication was found. The results suggest that in patients where such positive findings arise, periodic and intensive examinations should be performed, preferably by colonoscopy, to aid in the early detection of colon cancer.

5932 APPEARANCE AND GROWTH OF EARLY CARCINOMAS OF THE COLON-RECTUM. *(E.)* Ekelund, G.; Lindstrom, C.; Rosengren, J.-E. (Dep. Surg., Univ. Lund, Malmo, Sweden). *Acta Radiol. [Diagn.] (Stokh.)* 15(6):670-679, 1974.

A series of 30 patients with carcinoma of the colon-rectum was examined in retrospect for preceding signs of polypoid lesions at the site where a carcinoma had later been diagnosed. Repeated double contrast roentgenography had been used routinely for examinations of the colon during a mean observation time of 5 yr. When first demonstrated, all but 4 of the polypoid tumors had at least 1 roentgenographic sign of malignancy. The mean linear growth rate was 0.61 mm/month. The 5-yr survival rate was 57%. Metastases were found in 19 cases. The results suggest that tumors with roentgenographic appearance of malignancy should be removed as soon as possible and that careful observation with repeated roentgenography is imperative in the absence of malignant signs.

5933 PREOPERATIVE RADIOTHERAPY FOR COLORECTAL CANCER. *(E.)* Higgins, Jr., G. A.; Conn, J. H.; Jordan, Jr., P. H.; Humphrey, E. W.; Roswit, B.; Keehn, R. J. (VA Surg. Adjuvant Group, Washington, D.C.). *Ann. Surg.* 181(5):624-631, 1975.

In a prospective randomized trial, 700 patients with a confirmed histologic diagnosis of adenocarcinoma of the rectum or rectosigmoid were randomized to receive radiotherapy (2000-2500 rads in two weeks) prior to operation or to receive surgery alone. Five-year observed survival in the 453 patients on whom "curative" resection was possible was 48.5% in the x-ray treated group, compared with 38.8% in those who underwent surgery alone. In the 305 patients having low lying lesions which required abdominoperineal resection, survival in the group treated with x-ray was 46.9% compared with 34.3% in those who underwent surgery alone. Although these figures suggest a treatment benefit gained from the use of x-ray with surgery, neither figure is considered statistically significant. Histologically positive lymph nodes were found in 41.2% of the surgery only group but in only 27.8% of those patients receiving radiotherapy. Review of all patients who died during the study shows a consistently lower death rate from cancer in the radiotherapy group. Further studies are in progress to determine the optimal dose regimen.

5934 CARCINOID TUMOUR AND CROHN'S DISEASE. *(E.)* Tehrani, M. A.; Carfrae, D. C. (West. Infirm., Glasgow, Scotland), *Br. J. Clin. Pract.* 29(5):123-124, 1975.

A case of Crohn's disease is reported in which carcinoid tumor was an incidental finding on pathological examination of the resected small bowel. A tiny focus of carcinoid tumor (3-4 cells) was present in histological sections of the terminal ileum. Reexamination of the gross specimen demonstrated seven small yellow mucosal plaques in the terminal 30 cm of the small intestine. Sections confirmed the presence of multiple mucosal and subsequent mucosal deposits of carcinoid tumor, with no extension of the tumor through the bowel wall. A marked focal increase in argentaffin-cells was noted in the resected specimen. Only one previous case of carcinoid tumor coexisting with Crohn's disease has been reported in the literature. Although it is unlikely that a true relation exists between regional enteritis and carcinoid tumor other than the long-accepted association of malignancy and chronic inflammation, the possibility of coexistence should be kept in mind by both the clinician and the pathologist.

5935 VARIATIONS IN CARCINOEMBRYONIC ANTIGEN LOCALIZATION IN TUMORS OF THE COLON. *(E.)* Rogalsky, V. Y. (P. A. Herzen Res. Inst. Oncol., Moscow, USSR). *J. Natl. Cancer Inst.* 54(5):1061-1071, 1975.

The localization of carcinoembryonic antigen (CEA) in 78 tumors of the human colon was studied using antibody to CEA in an indirect Coons test. Morphologically it was possible to divide the neoplasms into two types: tissues in which CEA was localized in cells, and tissues in which malignant cells secreted CEA in greater or lesser quantity. In the first type of tissue, five forms of cells were noted: goblet-like, small vacuolar, limbic, those with intracellular accumulation of antigen, and diffuse. Tumor tissue secreting CEA could also be divided into 5 forms: holocrine-like, aprocrine-like, intraglandular, apical-basal, and lacunar-infiltrative. Thus, this study demonstrated that in malignant tissue there is a change in CEA localization as compared with that in cells of corresponding normal tissue. Several variations of CEA localization were observed. Tumor cells were found capable of secreting considerable amounts of this component. The change of antigen localization in the cell or its secretion was named antigenic translocation by the author.

5936 CHEMOTHERAPY AS AN ADJUVANT TO SURGERY FOR COLORECTAL CANCER. *(E.)* Lawrence, Jr., W.; Terz, J. J.; Horsley, III, S.; Donaldson, M.; Lovett, W. L.; Brown, P. W.; Ruffner, B. W.; Regelson, W. (Virginia Cancer Cent., Richmond). *Ann. Surg.* 181(5):616-623, 1975.

A combined intraoperative and postoperative adjuvant program of 5-fluorouracil (5 FU) for patients undergoing "curative" resection for adenocarcinoma of the colon and rectum was undertaken. Beginning in January 1968, patients were randomly assigned to an intraluminal 5 FU or intraluminal control (saline) group and were so treated at the time of surgical resection if findings at that time indicated that all gross neoplastic disease could be resected. Patients with operative findings denoting incurability were eliminated from the study after surgical exploration. Patients receiving intralumina 5 FU (30 mg/kg) received i.v. 5 FU (10 mg/kg) on each of the first 2 postoperative days and 5 subsequent postoperative courses of p.o. 5 FU (90 mg/kg in each 18-day course) over a 1-yr period. After a time of 6 yr, 156 patients had entered the study. Survival curves and "disease-free" curves for comparison of the group receiving 5 FU and the control group reveal no significant benefit from this intensive adjuvant course of 5 FU thus far. It is concluded that the benefit of prolonged adjuvant therapy with 5 FU for patients undergoing "curative" surgery for colorectal cancer is inadequate to justify its general use at this time.

5937 THE LATEST FINDINGS IN THE DIFFERENTIAL RADIOLOGICAL DIAGNOSIS OF ACUTE ILEUS. *(Ger.)* Bsteh, O. (Reg. Hosp., Mistelbach, Austria). *Radiol. Clin. Biol.* 44(1):14-23, 1975.

The pathophysiology of acute obstructive and paralytic ileus is considered, and the use of the barium enema to distinguish between these two conditions is discussed. In paralytic ileus, absorption, which ordinarily occurs in the right colon down to the splenic flexure, is replaced by exudation associated with gas production and motor paralysis. The left colon remains contracted, and the contrast medium passes readily into the right colon where it mixes with the formation of fluid levels. In obstructive ileus of the small intestine, the small intestine is distended with gas and shows fluid levels, while the colon is contracted throughout its entire length. Paralytic enterocolitis in elderly patients, which results from abnormal bacteriological flora in the small intestine, is characterized by discharge of toxic intestinal contents into the cecum where they produce inflammation and, in some cases, symptoms of appendicitis. In these patients, a barium enema provides a rapid and reliable diagnosis and often evacuates the intestine by dilating the spastically contracted left colon; cholinergic agents, administered parenterally, may also be indicated. A case report is presented for a patient in the 9th decade who developed acute ileus as a result of an extremely large scrotal hernia. A barium enema revealed paralytic ileus with enterocolitis, and the patient responded to conservative therapy. Barium enemas are also useful in the diagnosis of ileus in small children and of postoperative ileus. The technique is considered safe in all cases. In a 25-yr period, the author operated on 817 patients with perforated appendices with a total mortality of 4%; none of these patients died as a result of obstructive ileus. Cases of paralytic postoperative ileus were diagnosed and treated conservatively; none of these patients died either with the exception of 12 who also had peritonitis. Barium enemas are also recommended for establishing a differential diagnosis in rarer conditions, e.g., gallstone ileus, where the colon is empty and contracted, the small intestine is distended with fluid levels, and gas has collected in the biliary tract.

5938 BEHCET'S DISEASE AND TOXIC MEGACOLON. *(Ger.)* Roenspies, U.; Saegesser, F. (Dept. Surg., Univ. Lausanne, Switzerland). *Schweiz. Med. Wochenschr.* 105(7):199-204, 1975.

A 31-yr-old woman with a history of sporadic arthralgias in the fingers, wrists, and spine, an allergy to dust, and hayfever was treated for bilateral uveitis and allergic conjunctivitis; no evidence of hypopyon was found at this time. Two months later she was hospitalized with a fever, erythema nodosum, aphthae of the oral and vaginal mucosa, dermatographia, colic-like abdominal pain, and postprandial diarrhea. *Staphylococcus aureus* was cultured from the stools. X-rays revealed dilatation of the ileum and the onset of irregularities in the wall of the colon. After 1 week she developed rectal bleeding, meteorism, and marked dilation of the colon with loss of haustration. These clinical and radiological signs of toxic megacolon were consistent with a diagnosis of ulcerative hemorrhagic proctocolitis. However, this diagnosis was ruled out by normal endoscopic and histological findings. The patient responded to conservative treatment with transfusions, protein and electrolyte substitution, antibiotics, corticosteroids, salicylazosulfapyridine, and vitamins but developed a pulmonary embolism after one week. Ten days after the development of toxic megacolon, x-rays showed "thumb printing" of the en-

tire colon, as has been found in ischemic colitis; a double contrast enema showed colonic edema. A complete remission was obtained after 14 days of conservative therapy, but six months later a barium enema still revealed absence of haustration, particularly in the transverse and descending colon and the sigmoid, "thumb printing," and spicules. These findings are consistent with Crohn's disease or ischemic colitis. However, since the patient had no symptoms during the next 1.5 yr, Crohn's disease was ruled out. The history of bilateral uveitis, oral and vaginal aphthae, erythema nodosum, and pulmonary embolism suggests that the patient has Behcet's disease. Toxic magacolon is attributed to ischemic disorders in the entire colon associated with ulceration and a staphylococcal infection.

5939 CLINICAL ASPECTS, THERAPY, AND PROGNOSIS FOR CARCINOMA OF THE LARGE INTESTINE. *(Ger.)* Braun, L. (Surg. Clin. Polyclin., Univ. Munster, Germany). *Bruns Beitr. Klin. Chir.* 221(7): 503-515, 1974.

In a 22-yr period, 475 patients (266 males and 191 females, mean age 57.3 yr) were treated for histologically confirmed carcinomas of the colon, rectum, or anus. About half of all these tumors were located in the rectum, one-fourth to one-fifth in the sigmoid, and the rest occurred proportionately in the other parts of the large intestine. These patients consisted of 50% with stages A and B tumors, 17% with stage C, and 33% with stage D according to Duke's classification. Associated or pre-existing intestinal diseases consisted of hemorrhoids (15.6%), polyps (9.2%), typhoid fever (3.5%), diarrhea (3.1%), polyposis (2.9%), ulcerative colitis (2.6%), diverticulosis and/or diverticulitis (2.2%), fistulas and/or fissures (1.8%), periproctic abscess (0.7%), papilloma (0.4%), tuberculosis (0.4%), adenoma (0.2%), endometriosis (0.2%), and fibromatosis (0.2%). In the 390 cases for which information was available, 174 had metastases, 182 did not, and the presence or absence of metastases was in doubt in 34. The most common site of metastases was the lymph nodes (62 cases), followed by the peritoneum (43), liver (54), intestine (19), skin (16), lungs (10), female genitalia (9), bones (8), kidneys (4), and brain (2). Of these 457 patients, 423 (97.7%) were treated surgically. In 279 cases (66.0%) the tumor was completely removed; palliative surgery only was possible in 121 cases (28.6%); and tumors were inoperable at laparotomy in 23 cases (5.4%). Of the 457 operated patients, 102 (24.1%) died while still in the hospital; 12% died within the first 14 days after surgery. The causes of death were cardiovascular failure (31.4%), ileus (20.5%), inflammatory complications (19.6%), pulmonary embolism (16.7%), cachexia (8.0%), carditis (1.9%), and uremia (1.9%). Of the 16 patients treated conservatively, three lived for more than two yr. The five-yr survival rate was 0% for patients with inoperable tumors, 7.3% for those treated palliatively, and 32.6% for those who underwent radical surgery. The two-yr mortality was significantly lower (39.4%) in patients who received radiotherapy postoperatively than in those who did not (64.8%). After five yr, this difference was only 5.0%.

5940 A STUDY OF LYMPH NODES DRAINING COLORECTAL CANCER USING A TWO-STAGE INHIBITION OF LEUCOCYTE MIGRATION TECHNIQUE. *(E.)* Guillou, P. J.; Brennan, T. G.; Giles, G. R. (St. James Hosp., Leeds, England). *Gut* 16(4):290-297, 1975.

Leucocyte migration studies designed to investigate the sensitization of both peripheral and lymph node lymphocytes to tumor-associated antigens were performed in 14 patients suffering from adenocarcinoma of the colon and rectum. The results were compared with those of identical investigations on the peripheral blood and lymph node lymphocytes of ten control patients with benign gastrointestinal disease. Peripheral blood studies were performed on the morning of operation. Nodes were obtained from the root of the mesentery and from a paracolonic site in the controls and from the mesentery and a group of nodes draining the tumor in the patients; 14 nodes were obtained from the control subjects and 25 nodes from the patients with colorectal cancer. In five controls and 11 patients with cancer, a complete set of results was obtained for peripheral blood, mesenteric nodes, and paracolonic nodes. Leucocyte migration in the supernatant from control cells cultured with both normal colonic mucosal extract and tumor extract showed minor variations, but there were no significant differences between peripheral blood, mesenteric, and paracolonic lymph. The findings suggest that sensitization of local nodes may occur early in the progression of malignant disease; it may be lost when the lymph nodes become involved with tumor metastases. The results confirmed that some lymph nodes draining colorectal carcinomata contain sensitized lymphocytes, but the degree of sensitization to tumor extract does not seem greater than that seen in lymph nodes of the small bowel mesentery or of the peripheral blood.

5941 RESULTS OF THE COMBINED METHOD OF TREATMENT OF PATIENTS WITH CANCER OF THE RECTUM. *(Rus.)* Kozlovz, A. V.; Popova, T. V. (Dept. Clin. Radiol., Central Inst. Postgrad. Training Phys., Moscow, USSR). *Med. Radiol. (Mosk.)* 19(7):30-33, 1974.

5942 MACROFRACTIONATED PREOPERATIVE IRRADIATION IN COMBINED TREATMENT OF CANCER OF THE RECTUM. *(Rus.)* Sidorchenkov, V. O.; Baisogolov, G. D.; Berdov, B. A. (Dept. Radiother., Sci. Res. Inst. Med. Radiol., USSR). *Med. Radiol. (Mosk.)* 19(7):39-45, 1974.

5943 POLYPOSIS OF THE RECTUM AND COLON. *(Bul.)* Vijacki, L.; Krastev, M.; Manolov, G. (Fac. Med., Sofia, Bulgaria). *Khirurgiia (Sofiia)* 27(2): 147-151, 1974.

5944 CHANGES IN THE PHAGOCYTIC ACTIVITY OF LEUKOCYTES IN PATIENTS WITH ACUTE APPENDICITIS. *(Rus.)* Rybakov, A. I.; Dovzhikov, V. I. (Zaporozhie Med. Inst., USSR). *Vestn. Khir.* 114(2):42-44, 1975.

5945 A CASE OF PRIMARY CYSTIC PNEUMATOSIS OF THE COLON LOCALIZED TO THE SPLENIC FLEXURE. *(Fr.)* Mouchet, A.; Guivarc'h, M.; Marquand, J.; Barbagelata, M. (No affil.). *Chirurgie* 100(10):738-745, 1974.

5946 SURGICAL RESULTS IN POLYPOSIS OF THE COLON. *(Ger.)* Kugler, S.; Koch, G. (Dept. Gen. Surg., Univ. Clin. Surg., Hamburg-Eppendorf, Germany). *Bruns Beitr. Klin. Chir.* 221(8):583-591, 1974.

5947 MORPHOLOGICAL CHARACTERISTICS OF COLONIC POLYPOSIS. *(Ger.)* Jansen, H. H. (Inst. Pathol., Munic. Clin., Darmstadt, Germany). *Leber Magen Darm* 4(3):122-124, 1974.

5948 LYMPHOMAS OF THE INTESTINE--A CLINICOPATHOLOGICAL STUDY, WITH SPECIAL COMPARISON TO GASTRIC AND NODAL LYMPHOMAS. *(Jpn.)* Mari, S.; Yamaguchi, K.; Kino, I.; Oota, K. (Tokyo Univ. Med. Sch.). *Gan No Rinsho* 20(6):484-489, 1974.

5949 TREATMENT OF CARCINOMA OF THE RECTUM BY ELECTROCOAGULATION. *(Cs.)* Masurka, V.; Misek, J. (Fac. Med., Charles Univ., Hradec Kraklove, Czechoslovakia). *Rozhl. Chir.* 53(9):585-589, 1974.

5950 MALIGNANT TRANSFORMATION OF A PARARECTAL FISTULA CURED BY RADICAL SURGERY AND LOCAL PLASTIC REPAIR. *(Rus.)* Manuilenko, B. A. (Sakhalin Dist. Oncol. Clin., USSR). *Vestn. Khir.* 114(2):136-137, 1975.

5951 LONG-TERM RESULTS OF TREATING AN APPENDICEAL INFILTRATE. *(Rus.)* Brizgalov, V. E.; Firsov, V. D. (no affil.). *Sov. Med.* (2):100-103, 1975.

5952 RADIOLOGICAL DIAGNOSIS OF LATE COMPLICATIONS OCCURRING AFTER SURGERY ON THE RECTUM AND COLON. *(Rus.)* Siborov, V. S.; Chekurishvili, R. K. (Res. Lab. Proctol., Minist. Health RSFSR, Moscow, USSR). *Sov. Med.* (2):26-28, 1975.

5953 CONGENITAL MEGACOLON IN NEONATES AND INFANTS. *(Fr.)* Pasquie, M.; Petel, B. (Clin. Pediatr. Orthoped. Surg., Toulouse, France). *Cah. Med.* 15(10):669-676, 1974.

5954 GASTRECTOMY AND SURGERY FOR A HERNIA OF THE MESOCOLON ASSOCIATED WITH INTESTINAL MALROTATION. *(Ger.)* Woziwodzki, G. (Pritzwalk Dist. Hosp., Germany). *Zentralbl. Chir.* 99(45):1437-1438, 1974.

5955 SPONTANEOUS PERFORATION OF THE COLON AND ITS TREATMENT. *(Ger.)* Otto, K. (Med. Coll., Lubeck, Germany). *Zentrabl. Chir.* 99(44):1390-1394, 1974.

5956 PRECANCEROUS CONDITIONS OF THE COLON. *(Ger.)* Otto, P. (Med. Coll., Hannover, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:319-323, 1974.

5957 SEASONAL VARIATIONS IN THE CLINICAL COURSE OF ACUTE APPENDICITIS IN AREAS WITH HOT CLIMATES. *(Rus.)* Altiev, B. A. (Nebit-Dag Munic. Hosp., USSR). *Klin. Khir.* (3):71-72, 1975.

5958 CLINICAL SYMPTOMS AND RADIOLOGICAL FEATURES OF POLYPS IN THE APPENDICEAL STUMP. *(Ger.)* Keiler, A.; Staffen, A.; Zacherl, H.; Bardach, G. (2nd. Surg. Clin., Univ. Vienna, Austria). *Zentralbl. Chir.* 99(44):1402-1405, 1974.

5959 COMPLICATIONS OF DIVERTICULA OF THE COLON AND THEIR SURGICAL TREATMENT. *(Fr.)* Jacquet, N.; Desaive, C.; Melon, G.; Herman, P. (Surg. Clin., Univ. Liege, Belgium). *Rev. Med. Liege* 29(24):764-766, 1974.

5960 STATUS OF THE INTESTINE IN THYROID DISEASE. *(Rus.)* Mosin, V. I. (Stavropol Med. Inst., USSR). *Probl. Endokrinol.* 20(6):110-112, 1974.

5961 LYMPHANGIECTASIA IN THE MESOCOLON. *(Hun.)* Jakab, F.; Detky, B. (Clin. Surg. II, Semmelweiss Med. Univ., Budapest, Hungary). *Magy. Sebesz.* 27(6):401-404, 1974.

5962 COLON INTERPOSITION. *(Hun.)* Pas, E.; Pogany, Cs. (Clin. Surg. I, Semmelweiss Hosp., Budapest, Hungary). *Magy. Sebesz.* 27(6):382-384, 1974.

5963 SEVERE HEMORRHAGE IN DIVERTICULAR DISEASE OF THE COLON. *(It.)* Franchini, A.; Giardino, R. (1st. Semeiot. Chir., Univ. Bologna, Italy). *Chir. Gastroenterol.* 8(3):357-362, 1974.

5964 SINGLE POLYPS OF THE RECTUM--A PRECANCEROUS STAGE? *(Ger.)* Junemann, A.; Sailer, R.; Schacht, U. (Chir. Univ. Clin., Dusseldorf, Germany). *Med. Klin.* 69(43):1745-1748, 1974.

5965 ISCHAEMIC COMPLICATIONS OF OBSTRUCTIVE COLORECTAL CANCERS. *(It.)* Saegesser, F.; Tabrizian, M.; Rebagliati, M. (Serv. Univ. Chir., Lausanne, Switzerland). *Chir. Gastroenterol.* 8(3):264-276, 1974.

5966 DIFFERENTIAL DIAGNOSIS OF ACUTE APPENDICITIS. *(Rus.)* Pugleeva, V. P.; Mamedov, I. M.; Soloviev, V. A. (1st Leningrad Med. Inst., USSR). *Vestn. Khir.* 114(2):44-48, 1975.

5967 SIGMOID VOLVULUS ASSOCIATED WITH OTHER DISEASES. *(Rus.)* Stegailov, R. A. (Monchegorsk Assoc. Munic. Hosp., Murmansk District, USSR). *Vestn. Khir.* 114(2):131-132, 1975.

5968 DIVERTICULOSIS AND DIVERTICULITIS. *(Ger.)* Reifferscheid, M. (Med. Fac., Rhenish-Westphalian Tech. Coll., Aachen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:324-328, 1974.

5969 MALIGNANT TUMORS OF THE ANAL CANAL AND ANAL MARGIN. *(Fr.)* Pellet, B.; Saegesser, F.; Ozzello, L.; Vulliet, F. (Dept. Surg., Univ. Lausanne, Switzerland). *Schweiz. Med. Wochenschr.* 105(18):587-590, 1975.

5970 CARCINOID TUMORS OF THE VERMIFORM PROCESS. *(Rus.)* Sorinov, A. N.; Platonova, O. N. (Leningrad Pediatr. Med. Inst., USSR). *Vestn. Khir.* 114(2):48-50, 1975.

5971 ELECTRIC STIMULATION OF INTESTINAL MOTILITY IN THE POSTOPERATIVE PERIOD. *(Rus.)* Nechai, A. I.; Ostrovskaia, M. S.; Gudkov, G. V.; Baranovskii, A. L. (S. M. Kirov Mil. Med. Acad., Leningrad, USSR). *Vestn. Khir.* 114(1):108-112, 1975.

5972 THE SO-CALLED PRIMARY NECROSIS OF THE COLON: CONSIDERATIONS ON FIVE FURTHER CASES. *(Fr.)* Duveau, D.; Horeau, J. M.; Josso, A.; Morin, A.; Grolleau, J. Y.; Keribin, D.; Kropff, G. (Hosp. St. Jacques, Nantes, France). *Ann. Chir.* 28(12):1001-1008, 1974.

5973 TOXIC MEGACOLON. *(Fr.)* Friede, J.; Doyon, M.; Rousseau, B. (Cent. Hosp., Univ. Laval, Quebec, Canada). *Vie Med. Can. Franc.* 3(9):851-855, 1974.

5974 CARCINOMA OF THE TRANSVERSE COLON: A LATE-DIAGNOSED TUMOR. *(Fr.)* Roseau, E.; Kapron, A. M. (Hosp. Cochin, Paris, France). *Nouv. Presse Med.* 4(10):725-727, 1975.

5975 PERFORATION WITH MASSIVE MALENA [SIC] IN A CASE OF CARCINOMA OF COLON. *(E.)* Asraf, S. M.; Prakash, A.; Mital, R. V. (Maulana Azad. Med. Coll., New Delhi, India). *Indian J. Cancer* 11(4): 471-472, 1974.

5976 CONDYLOMA ACUMINATA AND ANAL CARCINOMA: TWO CASES OF PROBABLE MALIGNANT TRANSFORMATION. *(Fr.)* Vilotte, J.; Neuburger, P.; Sonsino, E.; Mesnier, J. M. (Hosp. Bichat, Paris, France). *Arch. Fr. Mal. App. Dig.* 63(7):595-602, 1974.

5977 TUMORS OF THE APPENDIX. *(E.)* Schmutzer, K. J.; Bayar, M.; Zaki, A. E.; Regan, J. F.; Poletti, J. B. (Queen Angels Hosp., Los Angeles, Calif.). *Dis. Colon Rectum* 18(4):324-331, 1975.

5978 PRIMARY LINITIS PLASTICA OF THE COLON: REPORT OF A CASE AND REVIEW OF THE LITERATURE. *(E.)* Chowdhury, J. R.; Das, K.; Das, K. M. (Nassau County Med. Cent., East Meadow, N. Y.). *Dis. Colon Rectum* 18(4):332-338, 1975.

5979 APPENDICITIS--STILL A POTENTIALLY LETHAL DISEASE. *(E.)* Hughes, J. H.; Kurtz, R. G. (Med. Coll. Ohio, Toledo). *Ohio Med. J.* 71(6):387-389, 1975.

5980 CLINICAL EXPERIENCES WITH COLONOSCOPY: A REVIEW OF 100 CASES. *(E.)* Dagradi, A. E.; Alaama, A.; Ruiz, R. (Long Beach VA Hosp., Calif.). *Am. J. Gastroenterol.* 63(5):408-413, 1975.

5981 CARCINOMA OF THE LEFT COLON AND RECTUM: A REVIEW OF 103 PATIENTS. *(E.)* Hernandez, A.; Morovati, S. S. (Wilmington Med. Cent., Md.). *Del. Med. J.* 47(6):312-315, 1975.

5982 ARE ANTICHOLINERGICS OF USE IN THE IRRITABLE COLON SYNDROME? *(E.)* Kvey, K. J. (R. Prince Alfred Hosp., New South Wales, Australia). *Gastroenterology* 68(5):1300-1307, 1975.

5983 TRAUMATIC PERFORATION OF THE SIGMOID COLON THROUGH SCHISTOSOMAL ULCERATIONS. *(E.)* Culliford, A.; Ibrahim, I.; Worth, M. H. (New York Univ. Med. Cent., N.Y.). *Am. J. Surg.* 129(6):705-708, 1975.

5984 DISEASES OF THE COLON AND RECTUM IN BOLIVIA. *(E.)* Rios-Dalenz, J.; Smith, L. B.; Thompson, T. F. (Hosp. Metodista, La Paz, Bolivia). *Am. J. Surg.* 129(6):661-664, 1975.

5985 ISCHEMIC COLITIS IN ESOPHAGEAL SUBSTITUTION: AN UNUSUAL AND LETHAL COMPLICATION OF COLON INTERPOSITION. *(E.)* Pradhan, D. J.; Ikins, P. M. (Upstate Med. Cent., Syracuse, N.Y.). *Am. Surg.* 41(7):427-428, 1975.

5986 A RADIOLOGIC STUDY OF TUBERCULOSIS OF THE ABDOMEN (GASTROINTESTINAL TRACT). *(E.)* Kolawole, T. M.; Lewis, E. A. (Dep. Radiol., Univ. Ibadan, Nigeria). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(2):348-358, 1975.

5987 ISCHEMIC COLITIS. RADIOLOGY AND PATHOPHYSIOLOGY. *(E.)* Wittenberg, J.; Athanasoulis, C. A.; Williams, Jr., L. F.; Paredes, S.; O'Sullivan, P.; Brown, B. (Boston City Hosp., Mass.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(2):287-300, 1975.

5988 VOLVULUS OF THE LARGE GUT. *(E.)* Dadoo, R. C.; Singh, W.; Sekhon, G. S.; Keswani, R. K. (Med. Coll., Rohtak, India). *Am. J. Proctol.* 26(3):69-76, 1975.

5989 RETROSPECTIVE COMPUTER ANALYSIS OF COLORECTAL CANCER SURGERY. *(E.)* Fitzgerald, P. A.; O'Grady, J. F.; McMahon, P. J.; Golden, M.; McMullin, J. P. (Dep. Surg., Univ. Coll. Dublin, Ireland). *Bull. Soc. Int. Chir.* 33(5-6):507-512, 1974.

5990 INTESTINAL POLYPS IN THE NIGERIAN AFRICAN. *(E.)* Williams, A. O.; Prince, D. L. (Dep. Pathol., Univ. Ibadan, Nigeria). *J. Clin. Pathol.* 28(5):367-371, 1975.

5991 VOLVULUS OF THE CECUM. *(E.)* Andersson, A.; Bergdahl, L.; Van Der Linden, W. (Dep. Surg., Univ. Umea, Ostersund, Sweden). *Ann. Surg.* 181(6):876-880, 1975.

5992 MASSIVE NECROSIS AND PERFORATION OF THE COLON IN AMEBIASIS. *(E.)* Gupta, S.; Sharma, C. L. N. (Inst. Med. Sci., Varanasi, India). *Ann. Surg.* 41(7):429-431, 1975.

5993 ENTEROGENOUS CYST OF THE COLON PRESENTING AS A RETROPERITONEAL TUMOR IN AN ADULT. *(E.)* Govoni, A. F.; Burdman, D.; Teicher, I.; Smulewicz, J. J. (Queens Hosp. Cent., Long Island, N.Y.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(2): 320-329, 1975.

5994 CONSTIPATION OF PROLONGED DURATION: CASE REPORT. *(E.)* Pelot, D.; Berk, J. E. (Dep. Med., Univ. Calif., Irvine). *Am. J. Gastroenterol.* 63(3):252-254, 1975.

5995 POLYPOID LESION OF CECUM. *(E.)* Kretschmer, K. P.; LaZerte, G. D.; Pass, B. R. (St. Francis Xavier Cabrini Hosp., Seattle, Wash.). *West. J. Med.* 122(4):334-337, 1975.

5996 PNEUMATOSIS CYSTOIDES INTESTINALIS. REPORT OF TWO CASES. *(E.)* Svensson, J. O.; Spangberg, A. (Sundsvall Hosp., Sweden). *Acta Chir. Scand.* 141(1):79-81, 1975.

5997 THE NITROBLUE TETRAZOLIUM (NBT) TEST AND WHITE BLOOD CELL COUNT IN PATIENTS WITH ACUTE ABDOMINAL PAIN, WITH SPECIAL REFERENCE TO ACUTE APPENDICITIS. *(E.)* Bjorksten, B.; Wahlby, L. (Univ. Hosp., Umea, Sweden). *Acta Chir. Scand.* 141(1):65-68, 1975.

5998 DIASTATIC RUPTURE OF THE CECUM IN OBSTRUCTING CARCINOMA OF THE LEFT COLON. RADIOGRAPHIC DIAGNOSIS AND SURGICAL IMPLICATIONS. *(E.)* Novy, S.; Rogers, L. F.; Kirkpatrick, W. (Univ. Texas System Cancer Cent., Houston). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(2):281-286, 1975.

5999 COLITIS CYSTICA PROFUNDA. PSEUDO-TUMOURS IN THE RECTUM. *(E.)* Mulder, H.; Te Velde, J. (Univ. Hosp., Leiden, Netherlands). *Radiol. Clin. Biol.* 43(6):529-539, 1974.

6000 COLON AND RECTAL CANCER IN THE YOUNG ADULT. *(E.)* Howard, E. W.; Cavallo, C.; Hovey, L. M.; Nelson, T. G. (Walter Reed Army Med. Cent., Washington, D.C.). *Am. Surg.* 41(4):260-265, 1975.

6001 COLONIC POLYPOSIS. *(E.)* Messinetti, S. (Dep. Surg. III Univ., Rome, Italy). *Rend. Gastroenterol.* 6(3):179-191, 1974.

6002 STERCORACEOUS AND IDIOPATHIC PERFORATIONS OF THE COLON. *(E.)* Huttunen, R.; Heikkinen, E.; Larmi, T. K. I. (Dep. Surg., Univ. Oulu, Finland). *Surg. Gynecol. Obstet.* 140(5):756-760, 1975.

6003 COPPER AND ZINC LEVELS IN THE BLOOD SERUM AND URINE OF BILHARZIAL HEPATIC FIBROSIS. *(E.)* Soliman, L.; El Safouri, S.; Megahed, G. M.; El Maola, G. (Dep. Biochem., Cairo Univ., Egypt). *Experientia* 31(3):280-281, 1975.

6004 SYNCHRONOUS LEIOMYOSARCOMA AND ADENOCARCINOMA OF THE RECTUM: REPORT OF A CASE AND REVIEW OF THE LITERATURE. *(E.)* Alfonso, A. E.; Hertz, R. E. (Mem. Hosp. Cancer, New York, N.Y.). *Dis. Colon Rectum* 18(3):228-232, 1975.

6005 RECTOSIGMOIDAL MUCOSAL PUCKERING AND DIVERTICULOSIS. *(E.)* Ritchie, J. A. (Nuffield Dep. Clin. Med., Oxford, England). *Dis. Colon Rectum* 18(3):221-227, 1975.

6006 COMPLICATIONS IN COLONOSCOPY. *(E.)* Smith, L. F.; Nivatvongs, S. (Nat. Naval Med. Cent., Bethesda, Md.). *Dis. Colon Rectum* 18(3):214-220, 1975.

6007 COLONIC PSEUDOPOLYPS IN ASSOCIATION WITH AMEBIC COLITIS. *(E.)* Berkowitz, D.; Bernstein, L. H. (Montefiore Hosp., Bronx, N.Y.). *Gastroenterology* 68(4):786-789, 1975.

6008 DRUG-INDUCED COLITIS MIMICKING AN ACUTE SURGICAL CONDITION OF THE ABDOMEN. *(E.)* Tedesco, F. J.; Anderson, C. B.; Ballinger, W. F. (Washington Univ. Sch. Med., St. Louis, Mo.). *Arch. Surg.* 110(5):481-484, 1975.

6009 BARIUM GRANULOMA OF THE RECTUM: AN UNCOMMON COMPLICATION OF BARIUM ENEMA. *(E.)* Lewis Jr., J. W.; Kerstein, M. D.; Koss, N. (Yale Univ. Sch. Med., New Haven, Conn.). *Ann. Surg.* 181(4):418-423, 1975.

6010 LYMPHOGRANULOMATOSIS OF THE RECTO-SIGMOID REGION: A RARE PRIMARY MANIFESTATION AND LOCALIZATION. *(Ger.)* Kuhlmann, H. W. (St. Jurgen's St. Central Hosp., Bremen, Germany). *Dtsch. Med. Wochenschr.* 100(11):553-554, 1975.

See also, 5622, 5623, 5658, 5671, 5679, 5692, 5698, 5768, 5775, 5799, 5868, 5870, 6061, 6099, 6204, 6395, 6520, 6522, 6552, 6558, 6575, 6577, 6590, 6591.

LARGE INTESTINE

Ulcerative Colitis

6011 ULCERATIVE COLITIS AND ANAPHYLACTOID PURPURA. *(E.)* Craig, R. M.; Ashman, M.; Feldman, S.; Hasegawa, J. (Northwest. Univ. Med. Sch., Chicago, Ill.). *Am. J. Dig. Dis.* 20(5):487-493, 1975.

A 21-yr-old male was hospitalized after an episode of bloody diarrhea and abdominal cramping. Two flat erythematous patches developed on his legs 6 months later which were termed "erythema marginatum." Recurrent erythematous eruptions occurred, occasionally associated with diarrhea and abdominal cramping. Ulcerative colitis was diagnosed. His history included several strep throats throughout his life which had been treated with penicillin. One month prior to hospitalization, his own physician put him on 16 mg methylprednisolone/day which was tapered to 4 mg/day. A proctoscopic examination after admission revealed multiple circumscribed erythematous lesions seen throughout the rectal mucosa. A biopsy of the rectum revealed small foci of mucosal hemorrhages and some acute inflammatory cells in the tunica propria mucosae. A skin biopsy revealed leukoclastic angiitis. Prednisone was administered at 30 mg/day, gradually tapering. During the 2 yr following this hospitalization, he had had exacerbations of his skin lesions, sometimes associated with watery or bloody diarrhea, so that the dose of prednisone had to be adjusted. During one of these episodes the patient was treated for possible tuberculosis and a lung biopsy was consistent with Loeffler's pneumonitis. The patient is now maintained on prednisone as an outpatient. This is the first case of well-documented ulcerative colitis occurring during exacerbations of anaphylactoid purpura. It is suggested that other patients with anaphylactoid purpura be carefully observed for evidence of ulcerative colitis.

6012 PULMONARY VASCULITIS AND ULCERATIVE COLITIS. *(E.)* Forrest, J. A. H.; Shearman, D. J. C. (R. Infirm., Edinburgh, Scotland). *Am. J. Dig. Dis.* 20(5):482-486, 1975.

A 37-yr-old man was admitted with a 6 week history of intermittent bloody diarrhea, tenesmus, malaise, anorexia, and a weight loss of 21 lb. Bilirubin was 2.0 mg/100 ml, SGPT 136 U/ml, alkaline phosphatase 120 King-Armstrong U/100 ml. The provisional diagnosis was ulcerative colitis and cholangitis. The patient became dyspneic at rest 2 days later and developed a cough with blood-stained sputum. His temperature rose to 39 C. Prednisolone (60 mg/day) abated the fever, and the dyspnea disappeared within 24 hr. His chest x-ray, which had shown a bilateral pulmonary shadowing, improved greatly. His liver tests remained abnormal. The SGPT and alkaline phosphatase did not improve with intermittent courses of antibiotics. Biopsies taken at colonoscopy showed ulcerative colitis. He was readmitted with mild diarrhea 1 1/2 yr later and a chest x-ray was normal. A few days later he became febrile with an unproductive cough and the chest x-ray showed bilateral pulmonary shadowing, which again resolved with prednisolone in 72 hr. At this time sigmoidoscopy showed a moderate proctitis. That the lung lesion responded promptly to corticosteroids, and that the lesion recurred, ruled out the diagnosis of subacute fibrosing alveolitis. In this patient, repeated cytomegalovirus titers were normal over a 2-yr period, and rectal and liver biopsies showed none of the pathologic lesions characteristically produced by that virus. Although fibrosing alveolitis has been reported in association with chronic active hepatitis, this patient's liver pathology is one of a pericholangitis secondary to ulcerative colitis. Although a lung biopsy was not performed, the most likely explanation for the patient's lung lesion is that he suffered two episodes of pulmonary vasculitis which on each occasion coincided with a relapse of his ulcerative colitis. It is suggested that there should be greater awareness that ulcerative colitis patients may develop pulmonary disease, either associated with their colitis *per se*, or secondary to salazopyrine.

6013 ESTIMATION OF CARCINOEMBRYONIC ANTIGEN IN ULCERATIVE COLITIS WITH SPECIAL REFERENCE TO MALIGNANT CHANGE. *(E.)* Dilawari, J. B.; Lennard-Jones, J. E.; Mackay, A. M.; Ritchie, J. K.; Sturzaker, H. G. (St. Mark's Hosp., London, England). *Gut* 16(4):255-260, 1975.

Carcinoembryonic antigen (CEA) levels in patients with colitis were investigated and the findings correlated with various clinical aspects of the disease. In particular, the question as to whether CEA levels could be correlated with the presence of carcinoma or of dysplastic epithelial changes without carcinoma was studied. A hospital control group was composed of 59 patients with the following diagnoses: idiopathic megacolon or Hirschsprung's disease (7), diverticular disease without inflammation (5), rectal prolapse (5), hemorrhoids (18), anal fissure (6), perianal hematoma (3), other anal conditions (4), and miscellaneous conditions (11). A second patient group consisted of 139 patients with uncomplicated ulcerative colitis, i.e., with-

out evidence of dysplastic change in the rectal mucosa or carcinoma. A third patient group consisted of 7 patients with ulcerative colitis and epithelial dysplasia. The last patient group contained 7 individuals (6 men and 1 woman, aged 24-63 yr) with established carcinoma in ulcerative colitis. In a group of 70 healthy controls, all individuals had CEA levels of less than 17.5 ng/ml. These results are different from those in the hospital control group where 8 of the 59 patients had values higher than 17.5 ng/ml with a range of from 1 to 36 ng/ml. Generally, there was little difference in the results for patients with colitis and those in the hospital control group. In the colitic patients no correlation could be established between the actual CEA levels and the age of the patient, the activity of the disease, or the extent of involvement. Only 1 patient among the 7 with dysplastic changes in the rectal mucosa had a CEA level above the values found in the hospital control group. One patient among the 7 with malignant change had a CEA level of 105 ng/ml; the other 6 patients, including one with recurrent growth and peritoneal metastases, all had CEA values within the range of the hospital control group. Thus, the results of this study show that CEA values in ulcerative colitis have no correlation with length of history, extent of disease, or degree of activity. It is unlikely that malignant change would be detected by this test earlier than by other methods of investigation. It is not recommended that routine CEA examinations be performed on patients with ulcerative colitis.

6014 CARCINOEMBRYONIC ANTIGEN IN PATIENTS SUFFERING FROM ULCERATIVE PROCTOCOLITIS. *(E.)* Mitchell, A. B. S.; Gill, A. M.; Orchard, R. T.; Parkins, R. A. (Prince of Wales's Hosp., London, England). *Am. J. Dig. Dis.* 20(5):407-417, 1975.

Carcinoembryonic antigen (CEA) was measured by radioimmunoassay in blood samples obtained from all 59 patients with ulcerative proctocolitis seen within a 4-month period. The characteristics of this group of patients were compared with reported epidemiological studies in this disease. The current patients had a similar sex ratio and age of onset as those in other studies, but the extent of their disease was more limited. Among these 59 patients, 11 had elevated circulating CEA values. One of the 11 had a colonic carcinoma and another was pregnant. Excluding these two patients, the overall prevalence of elevated CEA levels was 17.5%. The prevalence in ulcerative proctitis was 7.1%, and in colitis was 19.9%. The patients with elevated plasma CEA values were compared with remaining patients in relation to factors known to be associated with an increased propensity for the development of colorectal carcinoma complicating ulcerative colitis. There was no difference in mean age of the patients at disease onset, nor was there any difference in disease duration, extent, and control. A significant correlation was found, however, between elevated plasma CEA levels and the severity of the initial attack. Accordingly, a significantly greater proportion of those with normal CEA concentrations, as compared with those with elevated CEA concentrations, had suffered a mild onset of ulcerative proctocolitis. One patient with premalignant changes in the rectal mucosa had consistently normal concentrations of plasma CEA. There was no significant correlation between elevated plasma CEA values and disease activity. The mean age of the 2 groups of patients was similar. During follow-up periods of at least 18 months no carcinoma has manifested in any patient. The significance of the elevated levels of circulating CEA in patients who suffer from ulcerative proctocolitis and do not have evidence of carcinoma cannot be decided at present. The raised CEA levels can only be regarded as genuine false positives in the detection of colorectal carcinoma should this complication eventually be found with the same frequency among those with normal and those with elevated circulating CEA levels.

6015 CARCINOMA AND EPITHELIAL DYSPLASIA COMPLICATING ULCERATIVE COLITIS. *(E.)* Cook, M. G.; Goligher, J. C. (Gen. Infirm. Leeds, England). *Gastroenterology* 68(5):1127-1136, 1975.

The pathology of 19 specimens of carcinoma complicating ulcerative colitis was reviewed with particular attention given to the incidence of epithelial dysplasia. The 19 patients (aged 28-74 yr) had a history of colitis ranging from 3-34 yr in duration. The carcinomas were more frequently multiple, more evenly distributed in the large intestine, and much more often of atypical macroscopic appearances and of mucoid histological type than ordinary colorectal carcinomas, but the proportion of poorly differentiated tumors complicating ulcerative colitis was not as high as had been previously reported. Of these 19 patients, 26% are alive and well at least 5 yr after surgery. Unequivocal epithelial dysplasia was demonstrated in some part of the large intestine in 15 of 19 specimens with colitis carcinoma, but was also found in 4 of 14 specimens from a "control" series of patients with longstanding total colitis but without carcinoma. Therefore, the finding of dysplasia in a rectal biopsy of a patient with colitis is not a reliable guide to the presence of a frank carcinoma elsewhere in the intestine. Whether such a finding may indicate a predisposition to the development of a growth in the future cannot be determined from the data of this study. When present in colitis, epithelial dysplasia is often patchy in distribution and frequently spares the rectum even in patients with definite carcinomas; this makes a negative rectal biopsy particularly unreliable in deciding on the absence of a tumor or the lack of predisposition to its development. It is suggested that multiple biopsies from different parts of the colon as well as the rectum are desirable if mucosal sampling is to be employed as a screening test.

6016 MONOAMINE OXIDASE ACTIVITY IN PATHOLOGICALLY CHANGED TISSUE OF THE LARGE INTESTINE. *(Pol.)* Matuszewski, W.; Kalina, Z.; Kaminski, K.; Foremny, Z. (Inst. Biol.-Physiol., Silesian Med. Acad., Katowice, Poland). *Pol. Tyg. Lek.* 29(46): 1973-1974, 1974.

6017 LONG-TERM FOLLOW-UP OF PSYCHIC AND SOMATIC FACTORS IN PATIENTS WITH ULCERATIVE COLITIS. *(Ger.)* Feiereis, H.; Schroter, D. (Med. Coll., Lubeck, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:557-560, 1974.

6018 CLINICAL ASPECTS OF THE PHYSICIAN-PATIENT-RELATIONSHIP: A CASE OF ULCERATIVE COLITIS. *(Ger.)* Lohmann, R. (Med. Clin., Univ. Koln, Germany). *Leber Magen Darm* 4(3):93-98, 1974.

6019 DRUG THERAPY. SULFASALAZINE. *(E.)* Goldman, P.; Peppercorn, M. A. (Harvard Med. Sch., Boston, Mass.). *N. Engl. J. Med.* 293(1):20-23, 1975.

6020 NONSPECIFIC ULCERATIVE COLITIS. *(E.)* Tanga, M. R.; Prat, Y. J.; Ewing, J. B. (Ottawa Gen. Hosp., Canada). *Indian J. Surg.* 36(2): 68-71, 1974.

6021 LEUKAEMOID REACTIONS ASSOCIATED WITH FULMINATING ULCERATIVE COLITIS. *(E.)* Colvin, B. T.; Suggett, A. J.; Lancaster-Smith, M. J.; Fisher, D. J. H. (London Hosp., England). *Postgrad. Med. J.* 51(593):178-180, 1975.

6022 EVALUATION OF GUT MICROFLORA DURING ADMINISTRATION OF AN ELEMENTAL DIET IN A PATIENT WITH AN ILEOPROCTOSTOMY. *(E.)* Dickman, M. D.; Chappelka, A. R.; Schaedler, R. W. (US Naval Hosp., Philadelphia, Pa.). *Am. J. Dig. Dis.* 20(4):377-380, 1975.

6023 THE TREATMENT OF ULCERATIVE COLITIS WITH SULPHASALIZINE. *(E.)* Eastwood, M. A.; Das, K. M. (Western Gen. Hosp., Edinburgh, Scotland). *Br. J. Hosp. Med.* 13(2):142-144, 149, 1975.

See also, 5622, 5956, 5973, 6505, 6523, 6580, 6581, 6585.

PANCREAS

6024 ELECTROPHORETIC PATTERNS OF ALKALINE PHOSPHATASE ISOENZYMES IN HUMAN SERA WITH ABNORMALLY HIGH ACTIVITY, AND AN UNUSUAL BAND OBSERVED IN SERA OF PATIENTS WITH PANCREATIC CANCER. *(E.)* Cha, C. J. M.; Mastrofrancesco, B.; Cha, S.; Randall, H. T. (Rhode Island Hosp., Providence). *Clin. Chem.* 21(8):1067-1071, 1975.

The separation by electrophoresis of the isoenzymes of alkaline phosphatase on cellulose acetate membranes and an unusual alkaline phosphatase band observed in sera from patients with pancreatic cancer is reported. Alkaline phosphatase isoenzymes in sera were resolved into seven different bands: L_1, B_1, Pl, L_2, I_1, I_2 and Pa, in decreasing order of electrophoretic mobility. The slowest moving band (Pa) was observed in the sera of 16 patients, 15 with pancreatic cancer and one with hemochromatosis. Sera of 50 other patients with malignant or benign diseases did not show the Pa band. The Pa band was more heat labile than the liver isoenzyme (L_1). The behavior of the Pa band toward inhibitors (L-phenylalanine and L-homoarginine) was similar to that of L_1. Sera containing the Pa band exhibited a diffuse band in the region where isoenzymes of intestinal origin migrate; however, its heat stability and stereospecific inhibition were different from those of intestinal isoenzymes in sera showing no Pa band. It is suggested that further studies be carried out to determine whether the 'slow-moving' bands observed by different studies are identical, and to establish the diagnostic value and the cellular origin of the serum Pa band.

6025 ASSOCIATION OF POLYARTHRITIS, SUBCUTANEOUS NODULES, AND PANCREATIC DISEASE. *(E.)* Tannenbaum, H.; Anderson, L. G.; Schur, P. H. (Robert B. Brigham Hosp., Boston, Mass.). *J. Rheumatol.* 2(1):14-20, 1975.

A patient with polyarthritis, panniculitis, and pancreatic carcinoma was given metabolic and immunologic tests to find a relation between the joint, skin and pancreatic lesions. A 63-yr-old white male presented with migratory polyarthritis of 2 months duration. Tender skin nodules developed over the right tibia and both malleolar areas of the right ankle 2 weeks prior to admission. These nodules were indurated, warm, and erythematous. There was a history of granulomatous prostatitis and diabetes mellitus. Aspiration of the arthritic left knee yielded a clear serous fluid, but a second aspiration after 6 days yielded a thick creamy synovial fluid containing fat globules. Needle aspiration from the fluctuant subcutaneous tissue of a nodular lesion yielded a brown viscous fluid containing similar fat globules. Both this and the synovial fluid contained small, negatively birefringent, rectangular particles between the fat globules. Skin biopsy revealed a marked neutrophilic infiltrate involving subcutaneous tissue and deep dermis, with extensive focal liquifaction-type necrosis, focal chronic inflammation, scattered foam cells, and fibrinoid necrosis of small vessel walls. Liver biopsy revealed a metastatic undifferentiated tumor. The left knee effusion reaccumulated within 2 days, and new areas of synovitis and panniculitis appeared. Prednisone (60 mg/day)

controlled the arthritis but not the panniculitis. Chemotherapy with bleomycin, adriamycin, cyclophosphamide, oncovin, and prednisone for the tumor also resolved the panniculitis but the patient died shortly after. Autopsy revealed a well-differentiated acinar cell carcinoma in the tail of the pancreas. Lungs showed interstitial thickening with fibrosis and edema. The serum contained elevated lipase levels of 11.9-12.3 Cherry-Crandall U. The creamy joint fluid had a fatty acid of 26mg%, glycerol 41 mg%, and triglyceride 48mg%; there were 2122 M lactate/liter. Skin biopsy from a panniculitis area showed no immunoglobulins or complement. Lymphocytes failed to take up ^{3}H-thymidine when exposed to non-specific mitogens, tuberculin, or streptokinase/streptodornase, but did respond to *Candida albicans*. Blood immunoglobulins, gamma G subclasses, hemolytic complement, and complement components were normal. Radioimmunoassay for the carcinoembryonic antigen was positive (18.5 ng/ml). It is concluded that lipolysis and immunologic mechansims are not involved in the pathogenesis of this disease.

6026 IMMUNOGLOBULIN CHARACTERIZATION OF HUMAN PANCREATIC FLUID. *(E.)* Brasher, G. W.; Dyck, W. P.; Hall, F. F.; Spiekerman, A. M. (Scott White Clin., Temple, Texas). *Am. J. Dig. Dis.* 20(5): 454-459, 1975.

The concentration and stability of pure pancreatic fluid immunoglobulins were studied in two patients with traumatic pancreatic fistulae. Single radial diffusion demonstrated that the fluid of both patients contained detectable levels of IgG and IgA. IgD and IgM were found in 1/3 of the random samples in concentrations of 2.0-2.5 mg/100 ml, and 5-7 mg/100 ml, resp. One random specimen contained 216 U/ml IgE. One patient had 15 mg/100 ml IgG, 3.6 mg/ml IgA, 2.8 mg/100 ml IgM, and less than 0.9 mg/100 ml IgD. The IgG:IgA ratio ranged from 0.65-4.70. IgA molecular weight was estimated at 155,000 daltons by gel-filtration column chromatography. There was a progressive decay of all immunoglobulin fractions during storage at 4 C. By 10 days, no immunoprecipitates could be detected, while proteolytic activity had progressively increased. It is concluded that the five major immunoglobulins are present in human pancreatic fluid.

6027 EVALUATION OF BILIARY AND PANCREATIC FUNCTION IN PANCREOLITHIASIS: VALUE OF THE DETERMINATION OF γ-GLUTAMYL TRANSPEPTIDASE. *(E.)* Nakajima, S.; Noda, A.; Toda, Y. (Nagoya Hoden Eisei Univ. Sch. Med., Japan). *Arch. Intern. Med.* 135(6):829-831, 1975.

The pancreozymin-secretin test was used to determine γ-glutamyl transpeptidase, bile pigment, amylase concentrations and volume of duodenal aspirate. Patients with pancreolithiasis (10) and 10 controls had a triple lumen tube with two balloons, positioned fluoroscopically in the third portion of the duodenum after an overnight fast. A skin test with pancreozymin was performed at the beginning of each test. A quantitative duodenal aspirate was obtained for two 10 min control periods after the aspirate became alkaline. Then, 1 CHR U/kg pancreozymin was injected i.v. and three additional samples of aspirate were collected at 10 min intervals. Secretin (1 CHR U/kg) was then injected i.v., and six more 10 min samples were collected. Blood specimens were taken immediately before the hormone injections, and 30 and 60 min after the secretin. Routine liver function tests and drip infusion cholangiography in all patients showed normal results. The concentrations and outputs of γ-glutamyl transpeptidase in response to the two hormones were significantly greater in the patients than in the controls. There was no correlation between the enzyme and the bile pigment concentrations. Mean amylase outputs in pancreolithiasis patients were significantly less than those of controls, but secretion was not impaired in the form of the disease with bulky calculi in the head of the pancreas. Pancreozymin-secretin stimulation produced no increase above the normal levels of 40 mU/ml for serum γ-glutamyl transpeptidase and 200 U/100 ml for amylase, in the controls. In the patients, the provocative serum enzyme test was positive (rising to at least twice the initial values) in 3 for transpeptidase and in 1 for amylase. It is concluded that γ-glutamyl transpeptidase in posthormonal aspirates is significantly greater in patients than in controls.

6028 PANCREATIC CHOLERA (W.D.H.A. SYNDROME): HISTOCHEMICAL AND ULTRASTRUCTURAL STUDIES. *(E.)* Rambaud, J. C.; Galian, A.; Scotto, J.; Hautefeuille, P.; Matuchansky, C.; Modigliani, R.; Pessayre, D.; Bernier, J. J. (Hosp. St.-Lazare, Paris, France). *Virchows Arch.* [*Pathol. Anat.*] 367(1):35-45, 1975.

A detailed histochemical and ultrastructural study was made of a pancreatic tumor in a case of pancreatic cholera. A 38-yr-old woman presented with acute dehydration and anuria. There was a 7-yr history of recurrent diarrhea and a 2-yr history of five progressive quadriplegic attacks. Laboratory findings showed hypokalemia (1.8 mEq/liter), high fecal water and potassium losses (3270 ml/day and 160 mEq/liter/day, resp.), and basal gastric achlorhydria. Laparotomy showed a tumor of the body of the pancreas with multiple small metastases on the liver, peritoneum, and ovaries. The tumor was resected, and radioimmunoassay and bioassay showed large amounts of vasoactive intestinal peptide and a small amount of glucagon. The primary tumor, but not the metastases, contained large cysts lined in some places by small intestine-like epithelium and in other areas by cells similar to pancreatic duct cells. These exocrine-like structures were closely connected with endocrine cells, suggesting a mixed tumor. Apart from a few D cells, the endocrine tumor cells could not be identified by histochemical stains. Ultrastructurally the cells resembled gastric and duodenal D1 cells in that they contained small secretory granules (diameter less than 300 nm) and numerous cytoplasmic bundles of filaments. No intermediate cells containing both zymogen and endocrine-like granules were found in the tumor. Lymph

node, liver, and peritoneal metastases were composed exclusively of endocrine cells. Normal duodenal D1 cells have been reported to produce gastric inhibitory peptide, a substance structurally and biologically similar to the vasoactive intestinal peptide secreted by the tumor. Histological patterns were normal in the gastric, gallbladder, jejunal, ileal, and right and left colonic mucosa. This strongly suggests that a humoral agent (possibly vasoactive intestinal peptide) secreted by the pancreatic tumor is responsible for impaired intestinal function and abnormalities in gastric juice and gallbladder bile composition in pancreatic cholera.

6029 PANCREATIC SECRETION IN PERNICIOUS ANEMIA. *(E.)* Dreiling, D. A.; Greenstein, A. J. (Mt. Sinai Sch. Med., New York, N.Y.). *Am. J. Gastroenterol.* 63(6):495-497, 1975.

This report presents data from standard secretin tests performed on 257 control subjects, 19 patients with pernicious anemia, 67 patients with atrophic gastritis with achlorhydria, and 11 patients with Zollinger-Ellison syndrome. Gross inspection of the data from pernicious anemia patients indicates that 7 of the 19 secreted water and bicarbonate in the hypersecretory range. The increased secretion in pernicious anemia patients is consistent for flow, bicarbonate output, and amylase production. Patients with atrophic gastritis showed average responses similar to those with pernicious anemia. However, the increases in secretion in pernicious anemia and atrophic gastritis were not of the order of magnitude of those seen in Zollinger-Ellison syndrome and were not statistically significant. Preliminary observations on serum gastrin levels and their response to secretin indicated that levels are very high in pernicious anemia and atrophic gastritis. However, the gastrin is "Big Big" gastrin in contrast to the lower molecular gastrins found in Zollinger-Ellison syndrome. Moreover, the gastrin level in pernicious anemia and atrophic gastritis does not respond excessively to secretin as observed in the Zollinger-Ellison syndrome. These data suggest that the pancreatic parenchyma in pernicious anemia and atrophic gastritis may be responding to the hypergastrinemia of these conditions but at a minimal level consistent with the relatively inactive form in which the gastrin, "Big Big" gastrin, is secreted into the blood. Alternatively, the increased flow, bicarbonate output, and enzymes secretion may result from the synergism between the increased but relatively inactive serum gastrin and the endogenous secretin stimulus.

6030 VASCULARIZATION OF THE PANCREAS AFTER GASTRIC SURGERY. *(Ger.)* Barke, R.; Schulz, H. G. (Dep. Med., Karl Marx Univ., Leipzig, Germany). *Radiol. Diagn. (Berl.)* 15(1):13-20, 1974.

To study the effect of gastrectomy on vascularization of the pancreas, combined selective angiography of the celiac and mesenteric arteries was performed on four patients 2 to 6 yr after they had undergone Billroth II procedures for peptic ulcer. Three of these patients had undergone total orad antecolic resections with ligation of the right gastroepiploic artery close to the duodenum; in some cases the gastroduodenal or pancreaticoduodenal arteries were included. The left gastroepiploic artery was also ligated, so that only the left gastric artery supplied the gastric stump. Selective celiac angiography showed postoperative hypoplasia of the gastroduodenal artery in a 46-yr-old man who had undergone surgery 3 yr before for an ulcer of the anterior wall of the duodenum; the superior posterior and superior anterior pancreaticoduodenal arteries and the right gastroepiploic artery were not visualized. The transverse pancreatic artery was very small and the left gastric artery was relatively small. Combined selective celiac and superior mesenteric angiography in a 45-yr-old man, who had undergone gastrectomy 2 yr before for a recurrent duodenal ulcer, revealed a normal pancreaticoduodenal arcade due to careful ligation of the right gastroepiploic artery. In a 47-yr-old man who had undergone surgery 6 yr before for an ulcer of the anterior wall of the duodenum, combined angiography demonstrated some hypoplasia of the gastroduodenal artery; upper jejunal branches were drawn up from the superior mesenteric artery. Some branches of the pancreatic artery to the posterior body of the pancreas were also detected. The pancreaticoduodenal arcades were normal in a 35-yr-old man who had undergone a Billroth II gastroenterostomy with a retrocolic anastomosis 5 yr before, surgery for a gastrojejunocolonic fistula 2 yr before, conversion of the retrocolic into an antecolic anastomosis, repeated gastrectomy for an anastomotic ulcer 1 yr before, and resection for a perforated jejunal ulcer 6 months before. A large pancreatic artery, from which the transverse pancreatic artery originated, provided a very good blood supply to the head of the pancreas. The right hepatic artery originated from the proximal trunk of the superior mesenteric artery. In this case, a variation in the course of the right gastroepiploic artery apparently made a good pancreatic blood supply possible. It is concluded that disorders in pancreatic function in gastrectomized patients may be attributed to a decreased pancreatic blood flow.

6031 MORPHOLOGICAL PATTERNS OF PRIMARY NON-ENDOCRINE HUMAN PANCREAS CARCINOMA. *(E.)* Cubilla, A. L.; Fitzgerald, P. J. (Mem. Sloan Kettering Cancer Cent., New York, N.Y.). *Cancer Res.* 35(8):2234-2248, 1975.

6032 CYSTADENOMA OF THE PANCREAS. *(E.)* Didolkar, M. S.; Malhotra, Y.; Holyoke, E. D.; Elias, E. G. (Roswell Park Mem. Inst., Buffalo, N.Y.). *Surg. Gynecol. Obstet.* 140(6):925-928, 1975.

6033 IMMUNOLOGY, TUMOR MARKERS, AND PANCREATIC CANCER. *(E.)* Zamcheck, N. (Boston City Hosp., Mass.). *J. Surg. Oncol.* 7(2):155-165, 1975.

6034 CARCINOMA OF THE HEAD OF THE PANCREAS DEVELOPING IN A YOUNG WOMAN WITH A CHOLEDOCHAL CYST. *(E.)* Wood, C. B.; Baum, M. (Welsh Nat. Sch., Med., Cardiff, Wales). *Br. J. Clin. Pract.* 29(6):160-162, 1975.

6035 ISOLATED GASTRIC VARICES--A HINT OF PANCREATIC DISORDERS. *(E.)* Rosch, W. (Dep. Med., Univ. Erlangen, Nuremberg, Germany). *Endoscopy* 6(4):217-220, 1974.

6036 PANCREATIC PSEUDOCYST WITH HEMORRHAGE INTO THE GASTROINTESTINAL TRACT THROUGH THE DUCT OF WIRSUNG. *(E.)* Lam, A. Y.; Bricker, R. S. (Washington Univ. Sch. Med., St. Louis, Mo.). *Am. J. Surg.* 129(6):694-695, 1975.

6037 HEMOCOAGULATION IN PATIENTS WITH CANCER OF THE PANCREAS BEFORE AND AFTER OPERATIVE INTERVENTION. *(Rus.)* Zhukovskaia, E. S.; Itin, A. B. (Inst. Exper. Clin. Oncol. Acad. Med. Sci. USSR, Moscow, USSR). *Sov. Med.* (2):78-81, 1975.

6038 HETEROTOPIC PANCREAS. *(Fr.)* Maffioli, C.; Kuntz, J. L.; Bonnet, D.; Coudoux, P.; Geoffroy, H. (Med. Clin. B, Reims, France). *Maroc Med.* 54(584):599-601, 1974.

6039 THE CLINICAL VALUE OF RETROGRADE PANCREATOGRAPHY FROM A SURGICAL VIEWPOINT. *(Ger.)* Endsberger, G. (Surg. Clin., Stuttgart-Bad Cannstatt, Germany). *Med. Welt* 26(5):179-181, 1975.

6040 A CASE OF CYSTADENOMA OF THE PANCREAS WITH INTERESTING ANGIOGRAPHIC FINDINGS. *(Jpn.)* Shinohara, M.; Hanyo, F.; Kobayashi, S.; Yamada, A.; Hamano, K.; Fujimoto, A.; Muto, H.; Ohi, I.; Imai, M.; Takada, T. (Tokyo Women's Hosp., Japan). *Rinsho Hoshasen* 19(12):959-965, 1974.

6041 PANCREATIC HYPERSECRETION IN A CASE WITH HEMOCHROMATOSIS. *(It.)* Gaia, E.; Croce, G.; Sategna, S. (S. Giovanni, Battista Greater Hosp., Turin, Italy). *Minerva Gastroenterol.* 20(2):60-61, 1974.

6042 CHRONIC PANCREATITIS AND CARCINOMA OF THE PANCREAS. *(Ger.)* Mohr, P.; Ammann, R.; Largiader, F.; Knoblauch, M.; Schmid, M.; Akovbiantz, A. (Kanton Hosp., Zurich, Switzerland). *Schweiz. Med. Wochenschr.* 105(18):590-592, 1975.

6043 THE PHYSIOLOGY AND PATHOPHYSIOLOGY OF FAT DIGESTION, ESPECIALLY IN REGARD TO PANCREAS FUNCTION. *(Ger.)* Erb, W. (Cent. Intern. Med., Univ. Frankfurt/Main, Germany). *Fortschr. Med.* 93(12):623-626, 1975.

6044 ABSCESSED PANCREATIC PSEUDOCYST OF A RARE LOCALIZATION. *(Rus.)* Beliaev, A. A.; Shindiaikin, A. P. (2nd Surg. Hosp., N. V. Sklifosovskii Res. Inst. First Aid, Moscow, USSR). *Vestn. Khir.* 114(4):117-119, 1975.

See also, 5628, 5629, 5631, 5639, 5646, 5664, 5665, 5666, 5672, 5673, 5689, 5690, 5702, 5816, 5833, 5847, 5865, 6046, 6048, 6058, 6069, 6397, 6469, 6519.

PANCREAS

Pancreatitis

6045 HEREDITARY PANCREATITIS. NONSPECIFICITY OF AMINOACIDURIA AND DIAGNOSIS OF OCCULT DISEASE. *(E.)* Riccardi, V. M.; Shih, V. E.; Holmes, L. B.; Nardi, G. L. (Massachusetts Gen. Hosp., Boston). *Arch. Intern. Med.* 135(6):822-825, 1975.

A family with hereditary pancreatitis involving males in five generations and the initial proband (originally diagnosed at 11 months) is reported in order to emphasize the marked phenotypic variation found in this disease, the ability to make a diagnosis in an asymptomatic individual, and the nonspecific nature of any associated aminoaciduria. Hereditary pancreatitis was diagnosed in four cases, twice based on clinical and surgical findings, once on back pain and pancreatic calcifications, and once by laboratory and x-ray film studies. Suspected hereditary pancreatitis was diagnosed four times, once from old medical records, and in three cases from descriptions provided by family members. The autosomal dominant nature of the disease in this family was evident from multiple generation involvement and the male-to-male transmission. The morphine prostigmine test and the hypotonic duodenogram were helpful in establishing the diagnosis. On the basis of 24-hr urinary amino acid excretion rates available from other cases, and from those in this family who were available, it is concluded that the aminoaciduria previously associated with this disorder is coincidental or nonspecifically related to acute pancreatic inflammation. An increased risk for pancreatic carcinoma was emphasized by the concern for that complication in the proband's grandfather. However, counseling regarding the risk for developing pancreatic carcinoma in hereditary pancreatitis in clearly affected and especially asymptomatic individuals at risk cannot be done until more information is available.

6046 CLINICAL TRIAL OF CDP-CHOLINE AS A MEDICAMENT FOR ACUTE PANCREATITIS AND ITS RELATION TO PANCREAS AND LIVER DAMAGE IN ACUTE PANCREATITIS. *(E.)* Ozawa, K.; Kitamura, O.; Uchida, K.; Honjo, I. (Fac. Med., Kyoto Univ., Japan). *Bull. Soc. Int. Chir.* 33(5/6):483-489, 1974.

In an attempt to understand some of the basic mechanisms involved in the induced ischemic state, changes of oxidative phosphorylation of the pancreas in acute pancreatitis were studied. Pancreas and liver mitochondria from guinea pigs were prepared; oxygen consumption was measured polarigraphically and phosphorylative efficiency was measured simultaneously with oxygen utilization by following pH changes with a glass electrode. Spectrophotometric techniques were used to assay respiratory enzymes. The maximal phosphorylation rate in brain and pancreas mitochondria declined rapidly, as compared to liver, kidney and heart mitochondria, where the initial rate of phosphorylation remained almost constant for 10 min. In pancreas mitochondria, state 3 respiration (with glutamate as the substrate) declined rapidly, whereas that with succinate plus glutamate was slightly affected, leading to a rapid decrease in the glutamate to succinate ratio to less than 1/3 of the original value 5 min after ischemia. The decrease in the glutamate to succinate ratio paralleled the fall in the lipid acyl ester bonds and a concomitant rise in lysolecithin. Administration of 15 mg/kg CDP-choline to 53 patients with pancreatitis showed a 60% amelioration of symptoms. It is concluded that CDP-choline prevents mitochondrial derangement due to aging-induced activation of phospholipase A.

6047 USE OF A PERITONEAL LAVAGE IN THE DIAGNOSIS OF EXPERIMENTAL ACUTE PANCREATITIS. *(E.)* Machiedo, G. W.; Brown, C. S.; Lavigne, J. E.; Rush, Jr., B. F. (Martland Hosp., New Jersey Med. Sch., Newark). *Surg. Gynecol. Obstet.* 140(6):889-892, 1975.

Additional biochemical parameters for the recognition of acute pancreatitis are presented, and how they can aid in the diagnosis of the disease is discussed. Dogs (24) were divided into five groups. Under sodium pentothal anesthesia, those in the control group underwent no further manipulation; another group underwent laparotomy only; and dogs in the last three groups had induced pancreatitis (injection of bile into the pancreas), intestinal ischemia and duodenal perforation, resp. Analysis was made of serum and peritoneal lavage fluid in a dog from each group at 30-min intervals for 4.5 hr. Parameters which were significantly elevated in dogs with pancreatitis compared with other groups included fluid amylase, LDH, proteolytic activity and intestinal alkaline phosphatase and serum amylase. It is concluded that these biochemical differences in the lavage fluid, when considered together with the physical characteristics of the fluid, can significantly aid the clinician in arriving at the diagnosis of acute pancreatitis.

6048 PANCREATIC FUNCTION TEST IN PATIENTS WITH BILLROTH II RESECTION WITH THE AID OF AN ENDOSCOPE. *(E.)* Tympner, F.; Rosch, W. (Dep. Med., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Endoscopy* 6(4):245-257, 1974.

A new method is presented which allows a quantitative pancreatic function test in patients with Billroth II resection. A Lagerlof tube and an endoscope with prograde optics was introduced without premedication and the tube was placed into the afferent loop under visual control. The procedure was successfully applied in 12 patients in whom retrograde pancreatography was also attempted. Comparison of pancreatograms and pancreatic secretion after secretin-pancreozymin stimulation showed that the function test is superior to radiology. While retrograde pancreatography produced successful results in only seven of the patients, a clinical diagnosis was obtained in all cases with the use of the pancreatic function test. Although there is a certain amount of annoyance to the patient without premedication, it is concluded that this test allows the clinician to gather valuable information in patients likely to have chronic pancreatitis, and in whom other procedures such as retrograde pancreatography are unsuccessful.

6049 A PATHOGENESIS FOR ALCOHOLIC PANCREATITIS. *(E.)* Cameron, J. L.; Zuidema, G. D.; Margolis, S. (Johns Hopkins Med. Inst., Baltimore, Md.). *Surgery* 77(6):754-763, 1975.

To determine whether increased serum triglyceride levels secondary to alcohol ingestion might play an important intermediary role in the initiation of pancreatitis, patients with prior episodes of alcoholic pancreatitis associated with hypertriglyceridemia were admitted to a metabolic ward and studied. Hypertriglyceridemia was induced by dietary means, in the absence of alcohol, in an attempt to reproduce the symptoms of pancreatitis. During a quiescent period in their diseases, 12 patients with prior alcoholic pancreatitis were studied. By lipid feeding (316-894 g/day), significant hypertriglyceridemia (greater than 600 mg/100 ml) was induced in 11 of the 12. Seven of these developed abdominal pain similar to but not as severe as that experienced during prior attacks of pancreatitis. Of these seven, four developed serum amylase elevation, and of the remaining three, one had a serum lipase elevation and one a urinary amylase elevation. It is suggested, on the basis of these and previous data, that increased serum triglycerides act as important intermediaries in the pathogenesis of acute pancreatitis in some alcoholic patients.

6050 LACTOFERRIN IN THE DUODENAL JUICE OF PATIENTS WITH CHRONIC CALCIFYING PANCREATITIS. *(E.)* Estevenon, J. P.; Sarles, H.; Figarella, C. (Dig. Pathol. Res. Unit., INSERM, Marseilles, France). *Scand. J. Gastroenterol.* 10(3):327-330, 1975.

Since lactoferrin is present in the pancreatic juice of patients with chronic calcifying pancreatitis, but has not been found in normal juice, its

presence in pathological pancreatic juice was investigated for possible clinical significance. A technique for collecting human duodenal juice with a rubber tube and preventing its contamination with saliva is described. The presence of lactoferrin was investigated in the duodenal juice of 15 patients with chronic calcifying pancreatitis, 52 controls without evidence of pancreatic disease and nine cases of pancreatic disease other than chronic calcifying pancreatitis. Immunological methods were used to determine the presence of lactoferrin before and after an i.v. injection of cholecystokinin-pancreozymin (3 Crick-Harper-Raper U) plus secretin (1 CU). Tests for lactoferrin were positive in 11, negative in three, and inconclusive in one patient(s) with chronic calcifying pancreatitis. All nine patients with pancreatic disease other than chronic calcifying pancreatitis were negative for lactoferrin. Of the 52 controls, five were positive, six inconclusive, and 41 negative for lactoferrin. It is suggested that this test be used in the diagnosis of chronic calcifying pancreatitis.

6051 MAGNESIUM AND CALCIUM IN DUODENAL JUICE: SECRETION PATTERN IN CHRONIC PANCREATITIS AND IN PRIMARY HYPERPARATHYROIDISM. *(Ger.)* Baltzer, G.; Goebell, H.; Muller-Reinhardt, B. (Med. Clin., Phillipps Univ., Marburg/Lahn, Germany). *Klin. Wochenschr.* 52(2):74-79, 1974.

The secretion of magnesium (concentration and output) was measured in human duodenal juice before and after stimulation of the pancreas by secretin and pancreozymin (1 unit/kg) and compared with data for calcium. Subjects included 27 (12 women, 15 men, average age 49.9 yr) with normal pancreatic function; 23 patients (8 women, 15 men, average age 38.7 yr) with moderate and 22 patients (8 women, 14 men, average age 45.9 yr) with severe pancreatic insufficiency; and 14 (7 women, 7 men, average age 41.5 yr) with primary hyperparathyroidism. In all functional states of the normal pancreatic gland, magnesium and calcium were secreted in close positive correlation. The concentration was lowered after secretin. Pancreozymin increased both the concentration and the output of the two elements. The parallel secretion was also found, with few exceptions, in patients with pancreatic insufficiency. The secretion of both electrolytes was significantly reduced after pancreozymin with the lowered enzyme secretion. In primary hyperparathyroidism associated with normal pancreatic function a higher than normal concentration and output of magnesium and calcium after pancreozymin were measured. When enzyme secretion in these patients was disturbed, the secretion of the two electrolytes was reduced. In primary hyperparathyroidism the secretion of magnesium was more strongly stimulated by pancreozymin than that of calcium. The behavior of magnesium and calcium was no longer parallel in this case. Observations made in the analysis of enzyme fractions in five different phases of enzyme secretion in different phases of pancreatic stimulation suggest the possibility that the deviating behavior of magnesium is related to a preferred secretion of an enzyme protein.

6052 ALCOHOLIC HYALIN-LIKE BODIES FOUND IN THE PANCREATIC ACINAR CELLS AND NERVE CELLS OF THE BRAIN. *(E.)* Kojima, K. (Cent. Pathol., Kooseiren, Niigata, Japan). *Acta Pathol. Jpn.* 25(3):281-293, 1975.

The presence of alcoholic hyalin in pancreatic acinar cells and nerve cells of the caudate nucleus of the brain is reported. In a series of about 150 autopsy cases of alcoholics, hyalinoid body formation in acinar cells was demonstrated in 13. Of these 13, 2 also had hyalinoid bodies in the nerve cells of the caudate nucleus. Electron microscopy performed on the pancreatic tissue of 2 of the cases showed these bodies to have a fibrillar filamentous structure consisting of fine filaments in parallel alignment, and no limiting membrane. Each fibril was 65-379 Å in diameter. The hyalinoid bodies in the brain cells had the same fibrillar filamentous structure, with fibrils ranging from 53-155 Å in diameter. Some of the filaments showed periodicity of alternately arranged electron-dense particles of 130 Å and electron-lucent spaces of 50 Å. It is concluded that hyalinoid bodies found in pancreatic acinar cells and brain nerve cells of alcoholics greatly resemble the alcoholic hyalin in liver cells.

6053 IMMUNOLOGICAL PARTICIPATION IN CHRONIC CALCIFYING PANCREATITIS. *(E.)* Dani, R.; Antunes, L. J.; Ribeiro, J. E. F.; Nogueira, C. E. D.; Ribeiro, T. (Dep. Intern. Med., Fed. Univ. Minas Gerais, Belo Horizonte, Brazil). *Digestion* 11(5/6): 333-337, 1974.

Immunologic participation in chronic pancreatitis was studied in 12 patients with chronic calcifying pancreatitis and 20 healthy volunteers. Among the pancreatitis patients, 9 were alcoholic, 11 were diabetic, and 5 had steatorrhea; none was receiving steroids. Serum samples were studied by immunodiffusion, indirect immunofluorescence, and an anticomplementary technique; the percentage of peripheral blood lymphocytes was also determined. Compared with the controls, the mean serum concentrations of IgA and IgM were significantly elevated in the patients with chronic pancreatitis. One patient had autoantibodies against nuclear factors, smooth muscle, and IgG, 2 patients had autoantibodies to gastric parietal cells, and immune complexes were observed in 11 patients and none of the controls. The percentages of T-active and total T lymphocytes were significantly decreased in the pancreatitis patients, while the B lymphocyte levels were normal. These results suggest an immunologic participation in chronic calcifying pancreatitis, which might explain the persistence and progressive nature of the disease.

6054 AN ALTERNATE MECHANISM FOR THE FORMATION OF PROTEIN PLUGS IN CHRONIC CALCIFYING PANCREATITIS. *(E.)* Allan, J.; White, T. T. (Univ. Washington Sch. Med., Seattle). *Digestion* 11(5/6): 428-431, 1974.

To study the mechanism of protein plug formation in chronic calcific pancreatitis, protein precipita-

tion in the ductal pancreatic juice was studied in specimens obtained after surgery on patients with varying types of pancreatic disease. Two general types of precipitation phenomena were observed. Concentration-dependent precipitation occurred when specimens of inactive pancreatic juice secreted at concentrations greater than 3 mg/ml were chilled on ice or stored at 4 C. The fine precipitates thus formed could be redissolved by warming at 37 C. With specimens containing active chymotrypsin, insoluble flocculent precipitates were formed even from dilute solutions; these precipitates did not redissolve on warming. Based on these results, it is suggested that the etiology of plug formation and calcification in the small and large ducts of patients with chronic calcifying pancreatitis is due to a series of partial activations of zymogens in the ductules; this process, which proceeds by an unknown mechanism, results in precipitate formation in localized regions of the pancreas with later spreading throughout the gland. The precipitates not washed out by the newly-secreted juice would enlarge and calcify *via* the absorption of proteins and salts from the pancreatic juice.

6055 SERUM ANTIBODIES IN HUMAN PANCREATIC DISEASE. *(E.)* Lendrum, R.; Walker, G. (St. Mary's Hosp., London, England). *Gut* 16(5):365-371, 1975.

The presence of serum antibodies in human pancreatic disease was studied using immunofluorescent techniques. Serum samples from 61 patients with acute pancreatitis, 12 patients with relapsing acute pancreatitis, 14 patients with chronic pancreatic disease, 13 patients with pancreatic neoplasms, and 12 healthy adults were studied by indirect immunofluorescence using human pancreas as substrate; the positive sera were then retested after absorption with human RBC or human saliva. The positive sera were also tested against human pancreatic subfractions, human thyroid and stomach, rat liver and kidney, and rat and guinea pig pancreas. A reproducible pattern of immunofluorescence, consisting of a coarse granular appearance in the cytoplasm of the acinar cells, was obtained with the sera of 21 patients with acute pancreatitis, 3 of 381 patients without evidence of pancreatic disease, and 1 healthy adult. Absorption of the positive sera with A, B, or O blood cells or with saliva of the 3 Lewis phenotypes had no effect on the immunofluorescence reactions. The immunoglobulin classes of the antibodies in the positive sera were IgG in 1 patient, IgA in 7 patients, IgG and IgA in 13 patients, IgM in 1 control, and IgG in the other 3 controls. Complement fixation was demonstrated in only 1 of 15 positive sera. The positive sera gave no consistent or characteristic pattern when tested against human thyroid and stomach, and against rat liver and kidney; the results of the tests for species specificity were inconclusive. Tissue fractionation and absorption studies indicated that the antigen involved was located in the microsomal pancreatic fraction and that it may have been organ specific. The precise nature of the antigen is unclear, although it is unlikely that it is an enzyme.

6056 PANCREATITIS FOLLOWING INTESTINAL BYPASS FOR OBESITY. *(E.)* Sorrell, V. F.; Knight, D. H.; Burcher, S. K. (Middlemore Hosp., Auckland, Australia). *Aust. N. Z. J. Surg.* 45(2):163-167, 1975.

Of 24 patients operated on for massive obesity, 16 underwent end-to-end 10"-10" jejunoileal bypass with drainage of the excluded small bowel into the transverse colon. If the gallbladder was found abnormal at surgery, cholecystectomy was carried out. There were three patients (two men and one woman, aged 31 to 39 yr) who developed the severe complication of pancreatitis in the postoperative phase. One patient developed severe upper abdominal pain which was diagnosed as acute pancreatitis; he recovered over a 10 day period. By dietary modifications and the elimination of alcohol, he remained well over a 12-month follow-up. The second patient to develop this complication died. Autopsy revealed hemorrhagic pancreatitis and retroperitoneal necrosis in the remnant of pancreas, with severe fatty degeneration of the liver. There was chronic cholecystitis, and several multifacetted stones in the gallbladder. His bile had been lithogenic two weeks prior to death. The third patient developed acute pancreatitis 5 months after the bypass surgery. Conservative treatment was used. One month later a cholecystogram revealed a nonfunctioning gallbladder with stones; a cholecystectomy was performed. Liver biopsies at the time of bypass surgery revealed mild to moderate fatty changes in these patients, and palpation of the gallbladders at the initial surgery suggested they were normal. If it can be shown that this complication is directly related to the intestinal bypass procedure used and not to preexisting gallbladder disease, then it is suggested that this method be dropped in favor of a less drastic bypass procedure.

6057 HYPERLIPOPROTEINEMIA AND ACUTE PANCREATITIS. *(Sp.)* Carmena, R.; Ascaso, J.; Ferrando, J.; Fornes, J.; Gonzales-Bayo, E.; Bort, J. (Med. Fac., Valencia, Spain). *Rev. Esp. Enferm. Apar. Dig.* 45(3):269-280, 1975.

Case reports are presented for six patients (four women and two men, aged 23-63 yr) with acute pancreatitis associated with hyperlipoproteinemia. Acute pancreatitis was diagnosed on the basis of clinical symptoms and significantly increased serum amylase activities (four cases). In two patients, blood lipid levels were so high that they interfered with serum amylase determinations and false negative results were obtained; pancreatitis was diagnosed by increased urinary amylase activities in these two cases. Cholelithiasis was diagnosed in two patients; none were alcoholics. Familial hyperlipoprotenemia was present in three patients, two of whom were siblings. One female sibling had had five episodes of acute pancreatitis during the 4 yr previous; three of these episodes occurred during pregnancy. She was placed on a low-fat diet to prevent recurrences. Primary hyperlipidemia was diagnosed in four patients and secondary hyperlipidemia in two. The two patients with secondary forms had type V hyperlipidemia which disappeared 8 days after hospitalization. Determination of serum lipid fractions during the attack of

acute pancreatitis showed that all six patients had increased serum cholesterol, triglyceride, phospholipid, and total lipid concentrations; chylomicrons were detected in three cases. Electrophoresis revealed that hyperlipidemia was type IV in two cases, type V in three and type II B in one. In addition to acute pancreatitis and hyperlipoproteinemia (type V), one patient was obese and had a familial form of diabetes which had been diagnosed 9 yr previously; 1 yr before admission he had had a myocardial infarction and an episode of acute pancreatitis. All patients responded to conservative therapy (nasogastric aspiration, parenteral fluids, analgesics, broad-spectrum antibiotics, etc.).

6058 ON THE TREATMENT OF COMPLICATED PANCREATIC CYSTS. *(Rus.)* Forafonova, L. N.; Kuzmina, N. A. (Regional Hosp., Aktyu Ginsk, USSR). *Sov. Med.* (2):81-85, 1975.

6059 CALCAREOUS PANCREATITIS. *(Rus.)* Tuzhilin, S. A.; Neporent, M. I. (I. M. Sechenov First Moscow Med. Inst., USSR). *Sov. Med.* (2):91-96, 1975.

6060 COMPLICATIONS OF ACUTE NECROTIZING PANCREATITIS. *(Cz.)* Podzimek, A.; Manhal, J.; Kubicek, V.; Schuck, M. (Surg. Clin., Fac. Med., Charles Univ., Plzen, Czechoslovakia). *Sb. Lek.* 77(2):58-64, 1975.

6061 A RARE COMPLICATION OF PANCREATITIS: STENOSIS OF THE COLON. *(Sp.)* Cantallops, J. G.; GilGrande, L. A.; Hernandez-Guio, C.; Melero Calleju, E.; Miranda Baiocchi, R. (Jimenez Diaz Found., Madrid, Spain). *Rev. Clin. Esp.* 134(3):291-294, 1974.

6062 ACUTE PANCREATITIS IN OLD AGE. REVIEW OF 35 CASES. *(Sp.)* Segura, J. M.; Perez Jimenez, F.; Cano, J. M.; De Dios, J. F.; Muro, J.; Ortiz Vazquez, J. (La Paz Munic. Sanit. Soc. Secur., Madrid, Spain). *Rev. Esp. Enferm. Appar. Dig.* 45(4):399-408, 1975.

6063 NEW CONCEPT IN CONSERVATIVE AND SURGICAL THERAPY OF ACUTE PANCREATITIS. *(Ger.)* Schonborn, H.; Pross, E.; Olbermann, M. (Med. Clin. II, Univ. Mainz, Germany). *Internist (Berlin)* 16(3):108-115, 1975.

6064 THE EFFECT OF ELASTASE INHIBITORS ON THE COURSE OF HEMORRHAGIC PANCREATITIS. *(Rus.)* Levitskii, A. P.; Stefanov, A. V.; Veretennikov, A. L. (Izhevsk Med. Inst., USSR). *Patol. Fiziol. Eksp. Ter.* (6):57-60, 1974.

6065 THE EFFICACY OF TRASYLOL IN THE TREATMENT OF ACUTE PANCREATITIS. *(Ger.)* Haberland, G. L. (Pharmaceut. Res. Cent., Bayer AG, Wuppertal, Germany). *Med. Welt* 25(50):2112-2117, 1974.

6066 INTRA-CYSTIC HAEMORRHAGE: A COMPLICATION OF PSEUDOCYST OF THE PANCREAS. *(E.)* Cordero, O. C.; Khademi, M.; Lazaro, E.; Swaminathan, A. P. (New Jersey Med. Sch., Newark). *Br. J. Radiol.* 48(571):602-603, 1975.

6067 RENAL CYST CAUSING AFFERENT LOOP OBSTRUCTION AND ACUTE PANCREATITIS. *(E.)* Bubrick, M. P.; Hitchcock, C. R. (Hennepin County Gen. Hosp., Minneapolis, Minn.). *Am. Surg.* 41(7):440-443, 1975.

6068 APPRAISAL OF OPERATIVE TREATMENT FOR CHRONIC PANCREATITIS: WITH SPECIAL REFERENCE TO SIDE TO SIDE PANCREATICOJEJUNOSTOMY. *(E.)* Sato, T.; Saitoh, Y.; Noto, N.; Matsuno, K. (Tohoku Univ. Sch. Med., Sendai, Japan). *Am. J. Surg.* 129(6):621-628, 1975.

6069 REACTIVE DUODENAL CHANGES IN CHRONIC PANCREATITIS SIMULATING CONTIGUOUS SPREAD OF PANCREATIC CARCINOMA. *(E.)* Blackstone, M. O.; Mizuno, H. (Dep. Med., Univ. Chicago, Illinois). *Gastrointest. Endosc.* 21(4):184, 1975.

6070 CARDIOVASCULAR LESIONS IN PANCREATITIS. *(E.)* Tuzhilin, S. A.; Dreiling, D. A. (Mt. Sinai Sch. Med., New York, N. Y.). *Am. J. Gastroenterol.* 63(5):381-388, 1975.

6071 HEMORRHAGIC PANCREATITIS WITH DUODENOPANCREATIC FISTULA IN A RENAL HOMOGRAFT PATIENT. *(E.)* Peitzman, S.; Agarwal, B. N. (VA Hosp., Philadelphia, Pa.). *Am. J. Gastroenterol.* 63(5):420-422, 1975.

6072 ULTRASTRUCTURAL STUDIES OF HUMAN ACUTE PANCREATITIS. *(E.)* Taura, S.; Tsunoda, T.; Yoshino, R.; Akashi, M.; Ito, T.; Tsuchiya, R.; Takebayashi, S. (Nagasaki Univ. Sch. Med., Japan). *J. Clin. Electron Microsc.* 7(3-4):347-348, 1974.

6073 INDICATIONS FOR THERAPY WITH PROTEINASE INHIBITORS IN SURGERY. CLINICAL AND ANIMAL EXPERIMENTS. *(Ger.)* Eckert, P.; Riesner, K.; Doehn, M. (Univ. Hosp., Hamburg-Eppendorf, Germany). *Med. Welt* 25(51/52):2154-2157, 1974.

6074 SEROTONIN CONTENT IN EXPERIMENTAL PANCREATITIS. *(Rus.)* Bagratuni, B. E. (Yerevan Inst. Grad. Med. Studies, USSR). *Zh. Eksp. Klin. Med.* 15(2):30-34, 1975.

6075 SUCCESS WITH EARLY OPERATION IN ALTERING THE COURSE OF PANCREATIC INFLAMMATORY LESIONS. *(E.)* Haff, R. C.; Torma, M. J.; Capps, Jr., W. F. (Wilford Hall USAF Med. Cent., Lackland Air Force Base, Tex.). *Am. Surg.* 41(7):436-439, 1975.

6076 NEW PALPATION TECHNIQUE FOR THE PANCREAS TO DIAGNOSE CHRONIC PANCREATITIS. *(Rus.)* Bojanovicz, K. (Gastroenterol. Hosp., Med. Acad., Lodz, Poland). *Klin. Med. (Mosk.)* 53(1):83-86, 1975.

6077 CERTAIN FEATURES OF ACUTE POSTOPERATIVE PANCREATITIS. *(Rus.)* Napalkov, P. N.; Kiselev, A. I. (Leningrad Sanit. Hyg. Med. Inst., USSR). *Sov. Med.* (3):53-57, 1975.

See also, 5339, 5340, 5625, 5628, 5639, 5646, 5647, 5648, 5657, 5664, 5665, 5673, 6035, 6042, 6377, 6391.

LIVER AND BILIARY TRACT

6078 LIVER FAILURE AND DRUG METABOLISM. *(E.)* Andreasen, P. B.; Ranek, L. (Rigshosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 10(3):293-297, 1975.

The drug-metabolizing capacity of the liver (as measured by the metabolic clearance of antipyrine in patients with hepatic encephalopathy) was investigated in order to determine whether these patients have lower antipyrine clearance than non-encephalopathic patients with liver disease. Of 13 patients with hepatic encephalopathy, eight had acute hepatitis and five, cirrhosis. All subsequently developed stage III encephalopathy, and only one survived. A second group of patients comprised five persons with cirrhosis. Data for these first two groups of patients were compared to those obtained from 21 subjects without liver disease and 19 patients with biopsy-proven cryptogenic and alcoholic cirrhosis, all without signs of encephalopathy. The hepatic microsomal metabolism of antipyrine was measured in the 18 patients with encephalopathy before, during, and after hepatic encephalopathy. The antipyrine clearance measured in 13 patients during hepatic encephalopathy (4.6 ml/min) was significantly lower than that of five patients investigated 4 weeks before or after hepatic encephalopathy (9.6 ml/min). It is concluded that the clearance of antipyrine as a measure of quantitative liver function is substantiated by these data, and its use as a prognostic indicator of acute liver failure is suggested.

6079 ON THE DIAGNOSTICAL VALUE OF THE INDOCYANINE CLEARANCE IN CHRONIC DISEASES OF THE LIVER. *(E.)* Janev, P.; Zarzova, E. (I.P. Pavlov Higher Med. Inst., Plovdiv, Bulgaria). *Folia Med. (Plovdiv)* 16(3):157-161, 1974.

The diagnostic value of the indocyanine green clearance test was studied in 42 patients with diseases of the liver. Of these, eight had clinically decompensated liver cirrhosis; 18 had diffuse hepatic involvement (including chronic hepatitis, chronic cholangiohepatitis, and hepatic steatosis); five had primary and/or metastatic carcinomas; one had hyperbilirubinemia; four had cholecystic disease and six had other disease with no hepatic involvement. Of all patients tested for indocyanine green clearance, 29 (69%) were positive, as compared to 12% for SGOT and 3.8% for SGPT, for example, among other liver function tests performed at the same time. There was a close correlation between the gravity of the liver disease and the deviation in indocyanine green clearance indices, especially in cases of liver cirrhosis. The half-elimination probe and the elimination constant were established as the most sensitive indices. Due to the high diagnostic value of this test, the simple techniques necessary for its performance, and the lack of side effects, it is concluded that the indocyanine green clearance test should be ranked as one of the more valuable methods in the estimation of impaired liver function.

6080 ALOPECIA, ASCITES, AND INCOMPLETE REGENERATION AFTER 85 TO 90 PER CENT LIVER RESECTION. *(E.)* Starzl, T. E.; Putnam, C. W.; Groth, C. G.; Corman, J. L.; Taubman, J. (Univ. Colorado Sch. Med., Denver). *Am. J. Surg.* 129(5): 587-590, 1975.

The clinical course of a 19 yr-old female with a well-differentiated hepatoma who underwent 85-90% hepatectomy was presented. Hepatic tissue remaining after surgery consisted of a lateral portion of the left lobe. The patient was supported postoperatively by i.v. therapy including continuous albumin infusion (serum albumin>3 g/100 ml). The patient, however, developed ascites and subtotal alopecia within a week. At no time had the patient received cytotoxic chemotherapy. Daily low-grade fevers persisted for 2 months. Hyperbilirubinemia and depressed prothrombin time returned to normal about 1 month after surgery. The ascites was treated successfully with diuretics and the patient's hair eventually regrew. Serial 99mTc liver/spleen scans showed substantial but incomplete liver regeneration, which reached a maximum 6 months after resection. After 20 months, liver function was normal and there was no evidence of recurrent tumor.

6081 HEMANGIOMAS OF THE LIVER IN INFANTS: REVIEW OF DIAGNOSIS, TREATMENT AND COURSE. *(E.)* Slovis, T. L.; Berdon, W. E.; Haller, J. O.; Casarella, W. J.; Baker, D. H. (Columbia-Presbyt. Med. Cent., New York, N.Y.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(4):791-801, 1975.

Clinical and roentgenologic findings were presented for six infants who presented with benign vascular liver tumors between the first day and third month of life. All had high cardiac output or frank congestive heart failure. Presenting signs also included abdominal mass or enlarging liver and cutaneous hemagiomas. I.v. urography, with total body opacification, showed abnormal liver findings in 3/6. 99mTc liver scans were abnormal in four patients. The most useful diagnostic test was arteriography, which showed hypervascular lesions and early venous runoff in all subjects. Four patients, who were managed non-surgically with digitalis, steroids and radiotherapy (150-600 rads) or combinations of these regimens were alive after 3 months to 2 yr. One patient died of congestive heart failure prior to initiation of therapy. The sixth patient underwent surgical excision and is alive 7 yr later. The successful medical management of these patients suggest that this mode of therapy should be used initially in infants who present with high output state or congestive heart failure and who have early draining hepatic veins on arteriography. Patients not in heart failure and without early draining hepatic veins should have a liver biopsy to rule out hepatoblastoma.

6082 THE POLYMERIZED ALBUMIN AND ANTI-ALBUMIN AUTOANTIBODIES IN PATIENTS WITH HEPATIC DISEASES. *(E.)* Lenkei, R.; Mota, G.; Dan, M. E.; Laky, M. ("Victor Babes" Inst., Bucharest, Romania). *Rev. Roum. Biochem.* 11(4):271-276, 1975.

Antibodies against polymerized albumin were detected in sera from four patients with liver disease and autoimmune disorders. Anti-polymerized albumin antibody was also detected in 65-95% of sera from 404 patients with various hepatic diseases including acute, chronic active and persistent hepatitis and cirrhosis. There was no correlation between the presence of hepatitis B antigen and anti-albumin antibody although 27-55% of albumin-antibody-positive patients were also hepatitis B antigen-positive. Anti-polymerized albumin was more commonly present, however, in patients with impaired liver function. It is suggested that anti-polymerized albumin antibody may be a useful test for liver function but may also contribute to false positive results when testing for hepatitis B antigen in certain patients.

6083 A COMPARISON OF SCANS AND ANGIOGRAMS IN SELECTING PATIENTS WITH HEPATOMAS FOR HEPATIC LOBECTOMY. *(E.)* Gammill, S. L.; Takahashi, M.; Jingu, K.; Stumpe, W.; Font, R. (Univ. Tennessee Med. Sch., Memphis). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(3):522-530, 1975.

The comparative abilities of ^{198}Au or ^{99m}Tc liver scan and selective hepatic angiography to predict operability were studied in 35 patients with hepatoma. Liver scan and angiography agreed regarding operability *versus* inoperability in 27 cases. In 23 cases, which were determined to be inoperable by both studies, inoperability was confirmed at laparotomy or autopsy. Three of the four patients in whom tumors were judged operable had tumor confined to one lobe at survey or autopsy; the fourth subject had microscopic metastases in an opposite lobe. Liver scan and angiography did not agree with respect to operability in eight patients, five of which were predicted correctly by angiography owing to its greater sensitivity in detecting neovascularization. These results indicate that combined liver scanning and selective hepatic angiography may be useful in predicting operability with respect to the primary lesion in patients with hepatoma.

6084 PATHOLOGICAL LIVER TESTS IN PATIENTS ON A LOW CARBOHYDRATE DIET. *(Ger.)* Lutz, W. (No affiliation). *Wien. Med. Wochenschr.* 125(18): 292-295, 1975.

SGOT, serum bilirubin and total plasma protein and protein fractions were determined in 190 patients with chronic liver disease who were placed on a low-carbohydrate diet (maximum of 12 g) with no restriction on protein and fat intake. No drugs were prescribed and no measures were recommended, e.g. abstention from alcohol, which might affect laboratory findings. Before institution of the diet, SGOT values fell into three groups: (1) from 13 to 20 mU in 126 cases, (2) from 21 to 40 mU in 50 cases, and (3) over 40 mU in 14 cases. SGOT values in group one normalized after patients had been placed on the diet for an average of 2 weeks, while those in groups two and three approached the normal range in most cases during the unspecified observation period. Leucine aminopeptidase values, determined in 23 patients, decreased over a 15 month period from a mean of more than 28 mU to a mean of 14 mU; normal values (below 20 mU) were attained after an average of ten months. Serum bilirubin values, which were initially elevated in 16 patients with no other signs of liver disease, decreased somewhat more slowly when carbohydrates were restricted. Using a method sensitive to 0.8 mg/100 ml bilirubin, the mean initial value of 1.6 mg/100 ml decreased almost to normal values (mean 1.0 mg/100 ml) over a 50-month period. The low-carbohydrate diet produced no appreciable changes in total plasma proteins or protein fractions. Since γ-globulin levels increased and ascites became more pronounced, the low-carbohydrate diet had to be abandoned in three cirrhotics.

6085 PALLIATIVE IRRADIATION FOR LIVER METASTASES. *(E.)* Turek-Maischneider, M.; Kazem, I. (Acad. Hosp., Cathol. Univ., Nijmegen, Netherlands). *JAMA* 232(6):625-628, 1975.

The palliative effect of liver irradiation in patients with symptomatic liver metastases is reported. All 11 patients (aged 11-56 yr) presented with upper abdominal pain, a palpable liver, and abnormal liver scans. The treatment was delivered by a telecobalt therapy unit, using two parallel opposing fields to cover the entire liver. Field size ranged from 15 x 20 to 20 x 24 cm, with leading-off of corners to match the field-to-liver shape and to minimize bowel irradiation. A daily dose of 150 rads calculated in the midliver plane was administered 5 times/week for 2-3 weeks. Response to treatment was evaluated

according to the following categories: complete symptomatic relief, normalization or substantial improvement of liver function tests and liver scans (good); partial relief of symptoms, improvement of liver function tests, and reduction of the size of metastasis in the liver scans (moderate); and no symptomatic relief, no notable improvement of liver function tests, and no reduction in the size of metastasis in the liver scans (poor). Eight patients had good response; 2, moderate response; and 1 patient failed to respond to treatment. All patients tolerated the treatment and no deaths were attributed to liver irradiation. Three patients are alive at 4, 10, and 38 months, resp., after liver irradiation. It is concluded that radiation therapy is valuable for the treatment of symptomatic liver metastases, and is much safer than intrahepatic arterial infusion.

6086 THE HEPATORENAL SYNDROME: RECOVERY AFTER PORTACAVAL SHUNT. *(E.)* Ariyan, S.; Sweeney, T.; Kersten, M. D. (Yale Univ. Sch. Med., New Haven, Conn.). *Ann. Surg.* 181(6):847-849, 1975.

A case report is presented for a patient with the hepatorenal syndrome, a 78-yr-old man, admitted with cirrhosis of unknown etiology. On his first admission, he was noted to have tense ascites with 4+ pitting edema of the lower extremities. Hepatitis-associated antigen testing was negative; a liver scan showed a small liver with an enlarged spleen. He was placed on aldactone, digitoxin, a low salt and protein diet, and fluid restriction. Repeat liver biopsy showed fibrosis of the portal tracts. After several weeks, the ascites improved significantly, and he was discharged. One week prior to his second admission, he noticed increasing ascites and a 7-lb weight gain. Paracentesis revealed chylous ascites with 355 mg% lipids. Attempts at diuresis resulted in a rise in the blood urea nitrogen from 33 to 51. An enlarging umbilical hernia which was beginning to ulcerate was noted. Hepatic wedge pressure was 32 mm Hg; the i.v. cholangiogram was within normal limits and there was no hepatofugal flow. A successful side-to-side portacaval shunt was performed, and postoperatively, the hepatorenal syndrome was relieved. It is concluded that spontaneous recovery from the hepatorenal syndrome is rare; this patient is the 2nd case reported in the literature to recover from the hepatorenal syndrome following a surgical procedure to decompress the portal venous hypertension.

6087 ALPHA$_1$-ANTITRYPSIN DEFICIENCY AND HEPATIC CARCINOMA. *(E.)* Schleissner, L. A.; Cohen, A. H. (Harbor Gen. Hosp., Torrance, Calif.). *Am. Rev. Respir. Dis.* 111(6):863-868, 1975.

A patient with homozygous ZZ α_1-antitrypsin deficiency and severe emphysema, with a mixed carcinoma (hepatic and cholangiolar) of the liver in the absence of hepatic cirrhosis or fibrosis, was studied. When last admitted, the patient was in a respiratory coma. Serum alkaline phosphatase was 61.2 King-Armstrong U; neither serum α-fetoprotein nor hepatitis B antigen was detected, and there were no clinical signs of liver disease. On autopsy, the liver weighed 1,600 g and had a smooth capsular surface. The parenchyma was of usual consistency, displaying moderate passive congestion. Deep within the central portion of the right lobe was a firm, poorly delineated, whitish-tan mass measuring 6 cm in greatest diameter and surrounded by several smaller satellite lesions of similar description. Microscopic evaluation revealed hepatic parenchyma with normal ultrastructure. Intracellular inclusions in periportal hepatocytes were seen after periodic acid-Schiff stain. Malignant glandular structures were observed in the cholangiolar phase of the tumor. Because of the increasing frequency of hepatomas reported in patients with α_1-antitrypsin deficiency, it is suggested that a causal relation may exist.

6088 MANAGEMENT OF PRIMARY LIVER CELL CARCINOMA. *(E.)* Balasegaram, M. (Gen. Hosp., Kuala Lumpur, Malaysia). *Am. J. Surg.* 130(7):33-37, 1975.

An analysis of 352 cases of primary liver carcinoma is presented. The ratio of males to females was 4:1, and the patients ranged in age from 1 to 86 yr, with the majority aged 45-60 yr. Of the patients, 42 were admitted with acute abdomen after rupture of the hepatoma (15 of these died before any effective treatment could be carried out); 38 were admitted with bleeding esophageal or fundic varices associated with liver cirrhosis; and 52 had end-stage liver failure on admission. Of the total number of patients, 139 received no treatment: 34 left the hospital before treatment could be begun; 37 died of hemorrhage; and all 67 admitted with end-stage disease died within 2 weeks. Curative resection was attempted in 26 patients, and the results were: postoperative death (4); lost to follow-up (1); died within 1 yr (6); died within 2-3 yr (4); died within 3-4 yr (2); alive 2-3 yr later (4); and alive after 5 yr (5). Palliative treatment was attempted in the remaining 93 cases whose tumors were nonresectable. This included regional chemotherapy (25); complete hepatic dearterialization (60); and multiple dose therapy (60). Based on these data, it is suggested that attention be directed to detection of the definite causative factors of hepatic carcinoma, and perhaps more importantly, to the development of a good screening test to detect early carcinoma of the liver.

6089 ARTIFICIAL LIVER SUPPORT BASED ON HAEMOPERFUSION OF ADSORBENTS. *(E.)* Dunlop, E. H.; Weston, M. J.; Gazzard, B. G.; Langley, P. G.; Mellon, P. J.; Williams, R. (King's Coll. Hosp. Med. Sch., London, England). *Biomed. Eng. (NY)* 10(6):213-218, 1975.

The need for a device capable of performing the excretory functions of the liver during acute and chronic liver failure is discussed, and experience with an artificial liver support system is reviewed. Hemoperfusion through activated charcoal or Amberlite XAD-2 was tested in an animal model of acute liver failure. The model depends on surgically-

induced ischemia, and, as such, the liver necrosis was irreversible. Of 35 patients treated by charcoal hemoperfusion, all of whom were in deep hepatic coma from fulminant liver failure, 16 recovered consciousness completely and 13 are now alive and well at home 18 months later. It is concluded that although the technology of adsorbent hemoperfusion is at the moment in its infancy, the subject provides unique opportunities for bioengineers and clinicians to develop new therapeutic measures for the treatment of liver failure and to improve existing methods for kidney failure and poisoning.

6090 HEMANGIOSARCOMA OF THE LIVER. SPECTRUM OF MORPHOLOGIC CHANGES AND CLINICAL FINDINGS. *(E.)* Ludwig, J.; Hoffman, II, H. N. (Mayo Clin., Rochester, Minn.). *Mayo Clin. Proc.* 50(5):255-263, 1975.

Four cases of hemangiosarcoma of the liver and a brief review of the literature are presented, stressing features to enable the clinician and pathologist to make an earlier diagnosis. The patient group included 3 men and 1 woman, aged 36 (the woman) to 89 yr. The first case showed blood-filled cystic spaces in the liver lined by hemangiosarcoma cells. Vasoformative hemangiosarcoma tissue was seen to infiltrate the lumen of the hepatic vein. The final diagnosis was diffuse Kupffer cell carcinoma, of a nodular solid and microcystic type, with one small pulmonary metastasis, and ischemic hepatic necrosis; death was due to hepatic failure. The second case showed dilated hepatic sinuses lined by hyperchromatic hemangiosarcoma cells. The final diagnosis was a diffuse Kupffer cell sarcoma of the nodular and cystic type, without metastases, and possibly posthepatic cirrhosis. Death in this case was also due to hepatic failure. Hemangiosarcoma replacing the right lobe of the liver was seen in the 3rd case, with a small satellite nodule in the left lobe. The diagnosis was a circumscribed, nodular solid, and microcystic type of Kupffer cell sarcoma. Death was due to bronchopneumonia. Histologic examination of the liver in the 4th case showed multiple blood-filled cysts. An autoradiograph showed the typical α-ray tracks from thorium dioxide deposit (this patient had received 20 ml of thorotrast into the right carotid artery). Final diagnosis was a low-grade Kupffer cell sarcoma, of the diffuse cystic type, with thorium dioxide storage in the liver and lymphatic organs. Death came from exsanguination of the hepatic tumor. Analysis of these cases and a review of the literature indicated that these tumors are either predominantly cystic and fairly well-differentiated or are more solid and poorly differentiated. Well-differentiated tumors may resemble peliosis hepatitis or other benign conditions. It is concluded that the concept of Kupffer cell sarcoma is weakened by the fact that primary and secondary hemangiosarcomas in the liver, including their sinusoidal spread, are histologically indistinguishable.

6091 HEPATOCELLULAR CARCINOMA. A CLINICAL AND PATHOLOGICAL STUDY OF 33 CASES. *(E.)* Tsakraklides, V.; Templeton, A. C.; Estensen, R.; Good, R. A. (Dep. Pathol., Univ. Minnesota, Minneapolis). *Minn. Med.* 58(6):450-453, 1975.

An analysis of 33 cases of hepatocellular carcinoma seen at postmortem at the University of Minnesota Hospitals is presented; the histological features, metastatic patterns, diagnostic problems, and relation to cirrhosis are discussed. Of 12,070 case records examined, 33 hepatocellular carcinomas were found, comprising 0.27% of the autopsies recorded in an 18-yr period. All were caucasian, and 73% were male, with the peak incidence in the 6th, 7th, and 8th decades. The representing signs and symptoms were abdominal pain and discomfort in 15 cases, hepatomegaly in 6, jaundice in 5, and nausea and vomiting in 4. Hematemesis was the presenting symptom in 3, fatigue and weight loss in 2, and fever of unknown etiology in 2. The diagnosis was prior to death in 16 cases, and only at autopsy in the remaining 17. The average duration from onset of symptoms to death was 5.5 months; the longest interval was 18 months. Death was caused by gastrointestinal bleeding in 10 cases, hepatic coma in 9, pneumonia in 7, hepatorenal failure in 5, and was operative or postoperative in 2. Cirrhosis was an associated disease in less than half of these patients. The incidence of hepatocellular carcinoma appeared in this geographical area to be rising slowly, and the incidence of cirrhosis as well as the incidence of liver carcinoma in cirrhotics did not significantly increase. The data suggest that the patients at particular risk were elderly males with coarse nodularity of the liver.

6092 ENZYME REPLACEMENT IN NIEMANN-PICK DISEASE BY LIVER HOMOTRANSPLANTATION. *(E.)* Daloze, P.; Corman, J.; Bloch, P.; Delvin, E. E.; Glorieux, F. H. (Notre Dame Hosp., Univ. Montreal, Canada). *Transplant. Proc.* 7(1):607-610, 1975.

The feasibility of long-term enzyme replacement by liver homotransplantation as evaluated in a recipient afflicted in Niemann-Pick disease type A is reported. Hepatomegaly in an otherwise healthy 4-month old boy led to the early diagnosis of Niemann-Pick disease that was later confirmed by the demonstration of sphingomyelinase deficiency in WBC and cultured skin fibroblasts. Psychomotor retardation and massive hepatosplenic involvement were evident at age 1 yr. Orthotopic liver transplantation was performed at age 2 yr. Muscular activity increased, expression of the face became more mobile, and seizures that were frequent in the early postoperative period slowly became scarcer without specific medication. After transplantation, sphingomyelin did not reaccumulate in the grafted liver and could not be detected in urine. Indirect evidence appears to indicate that lipid deposits are beginning now to regress in some tissues, such as lungs and retina. Sphingomyelinase has been detected not only in the graft but also in several physiologic fluids including the cerebrospinal fluid. These observations raise the possibility for stabilization or even cure of some congenital enzymatic deficiencies by organ transplantation, when the deficient enzyme is not available by other means.

6093 REYE'S SYNDROME: EPIDEMIOLOGIC AND VIRAL STUDIES, 1963-1974. *(E.)* Linneman, Jr., C. C.; Shea, L.; Partin, J. C.; Schubert, W. K.; Schiff, G. M. (Univ. Cincinnati Coll. Med., Ohio). *Am. J. Epidemiol.* 101(6):517-526, 1975.

The experience with Reye's syndrome in a single medical center from 1963-1974 is reported; only those cases shown to have fatty liver were included in the study. During this time period, 58 children with Reye's syndrome confirmed by liver biopsy were treated at this center. Cases were clustered in the winter and spring with the peak in February and March. These coincided with the occurrence of influenza and, numerically, were associated more closely with influenza B than with influenza A. Of the 58 cases, 6 were associated with chickenpox. From 1963-1971, before the beginning of a systematic epidemiologic and virologic study, 26 children with Reye's syndrome were seen. Viral infection was documented in either the patient or a contact in only 19% of those studied. Between 1971-1973, after the study was initiated, 16 cases of Reye's syndrome were diagnosed and viral infection was confirmed in 56% of these. In 1974, an epidemic of Reye's syndrome occurred during an influenza epidemic and viral infection was found in either the patient or a contact in 81% of 16 cases. During this study no significant environmental toxic exposures could be identified. Most children had taken aspirin and other medications, and 7 children had a history of excessive aspirin ingestion. This study demonstrates that an association with viral infection can be proven in the majority of cases of Reye's syndrome when an intensive investigation is undertaken.

6094 ALPHA-1-FETOPROTEIN AND THE HEAT STABLE ALKALINE PHOSPHATASE IN SOME LIVER DISEASES. *(E.)* Zizkovsky, V.; Kordac, V.; Stepan, J.; El'gort, D. N. (Fac. Gen. Med., Charles Univ., Prague, Czechoslovakia). *Acta Hepatogastroenterol. (Stuttg.)* 22(2):92-97, 1975.

The presence of α_1-fetoprotein, heat-stable alkaline phosphatase, and Australia antigen (HB_sAg) was examined in 103 patients with porphyria cutanea tarda, 300 patients with cirrhosis, and 18 patients with primary liver carcinoma. Heat-stable alkaline phosphatase was detected in 46% of patients with porphyria cutanea tarda and in 61% of patients with primary liver carcinoma. α_1-Fetoprotein was detected in 61% of patients with primary liver carcinoma and in 2 patients with porphyria cutanea tarda in whom primary liver carcinoma was later proved. The simultaneous occurrence of α_1-fetoprotein and heat-stable alkaline phosphatase was found in 50% of cases with primary liver carcinoma. Neither the patients with porphyria cutanea tarda nor those with cirrhosis were HB_sAg-positive. HB_sAg could be detected in only 1 patient with α_1-fetoprotein-positive carcinoma of the liver. The finding of the heat-stable alkaline phosphatase in the blood sera of 46% of patients with porphyria cutanea tarda supports the hypothesis that this disease is a possible precancerous state. However, since the detection of the heat-stable alkaline phosphatase may be caused by the enzyme stabilized with one of the metabolites originating in porphyria cutanea tarda, the question requires further research.

6095 PATHOLOGY OF THE LIVER IN REYE'S SYNDROME. *(E.)* Svoboda, D. J.; Reddy, J. K. (Univ. Kansas Med. Cent., Kansas City). *Lab. Invest.* 32(5):571-579, 1975.

Case reports are presented for 6 patients (4 men and 2 women, aged 4-16 yr) with Reye's syndrome, of whom 4 died. Needle liver biopsy specimens were obtained from all patients either shortly before death or during recovery from several hours of coma. Fixed and stained portions were examined under the light and electron microscopes. Light microscopy revealed that inflammation and necrosis of liver cells were especially prominent in fatal cases. Contrary to previous reports, electron microscopy indicated that aside from a loss of matrix dense granules, alterations in mitochondrial structure were minimal or absent. Although an increase in the number of liver cell microbodies has previously been reported, no such increase was apparent in the present study. The most unusual ultrastructural change in microbodies in the present study was the presence of noncrystalline cores in the matrix. No ultrastructural features distinguished patients who died from those who survived. Although derangement of mitochondrial function may be important, it is concluded that such derangement in the pathogenesis of Reye's syndrome is not necessarily reflected in mitochondrial ultrastructure.

6096 GLUCOSE INTOLERANCE AFTER MASSIVE LIVER RESECTION IN MAN AND OTHER MAMMALS. *(E.)* Ida, T.; Ozawa, K.; Honjo, I. (Fac. Med., Kyoto Univ., Japan). *Am. J. Surg.* 129(5):523-527, 1975.

The relation between changes in glucose tolerance and in mitochondrial phosphorylative activity was studied after massive liver resection. In rabbits 24 hr after hepatectomy, when the phosphorylative activity in the mitochondria of the remnant liver was enhanced maximally, the blood glucose level did not increase significantly after glucose administration (2 g/kg). When the phosphorylative activity decreased to a submaximal level 48 hr after hepatectomy, the blood glucose level increased gradually but without a return to normal. About 6 weeks after partial hepatectomy, mitochondrial function gradually returned to normal levels and the glucose tolerance test results returned to within normal limits. The changes in the glucose tolerance tests in rabbits were also observed in Wistar rats and in a 27-yr-old man hepatectomized for blunt abdominal trauma. The return to normal of the glucose tolerance test results, however, was more rapid in rats: 3 hr after partial hepatectomy, the fasting blood glucose was at a hypoglycemic level; after 24 hr, the level increased to more than 150 mg/100 ml; and after 3 days, the glucose tolerance patterns returned to normal. The return of test results to normal in the patient was remarkably delayed: p.o. glucose

tolerance testing, 50 g, was carried out at 14, 44, and 90 days postoperatively; values did not return to normal until the second and third tests. Based on these data, it is suggested that the glucose tolerance test is very useful in evaluating the stage of regeneration in the liver remnant after massive liver resection.

6097 CORTISOL METABOLISM IN CHRONIC LIVER DISEASE. *(E.)* McCann, V. J.; Fulton, T. T. (R. Victoria Hosp., Belfast, Northern Ireland). *J. Clin. Endocrinol. Metab.* 40(6):1038-1044, 1975.

To further evaluate the effects of impaired degradation of cortisol, various parameters of cortisol metabolism were measured in 39 patients (22 men and 17 women, aged 13-74 yr) with chronic liver disease. These parameters included total and nonprotein-bound plasma cortisol measured as 11-hydroxycorticoids at intervals during the day, urinary excretion of cortisol, corticosteroid-binding globulin, and cortisol excretion rate. The group of patients included persons with active chronic hepatitis (9), persistent hepatitis (2), cryptogenic or postnecrotic cirrhosis (15), and alcoholic cirrhosis (13). The mean plasma total 11-hydroxycorticoids (11-OHCS) was lower in patients than in controls, although the difference was not statistically significant, and the normal circadian rhythm was maintained. However, in patients with chronic liver disease, a greater proportion of the 11-OHCS was in the nonprotein-bound state, resulting in an elevation of this fraction. Corticosteroid-binding globulin was lower in patients than in controls, although the difference was not statistically significant. Urinary cortisol excretion was significantly reduced, as was excretion of 17-ketosteroids. The cortisol secretion rate was normal. The elevation of nonprotein-bound 11-OHCS represents a resetting of the pituitary-adrenal feedback mechanism in these patients. It is suggested that elevation of plasma nonprotein-bound 11-OHCS, resulting from impaired metabolism and reduced protein binding of cortisol in patients with hepatic disease, may explain the features of endogenous hypercorticsm seen in such patients. Moreover, in the presence of impaired steroid metabolism and reduced protein binding, these patients may exhibit an increased sensitivity to corticosteroid therapy, and therefore administration of a reduced dosage may be advisable.

6098 LIVER PATHOLOGY IN ALPHA$_1$-ANTITRYPSIN DEFICIENCY: A REVIEW. *(E.)* Cohen, C. (South African Inst. Med. Res., Johannesburg). *S. Afr. Med. J.* 49(21):849-852, 1975.

The association of liver disease with alpha$_1$-antitrypsin (AAT) deficiency (AATD) is reviewed. The lowest serum levels of AAT, a low-molecular weight glycoprotein synthesized in the liver, are found in persons with the homozygous Z phenotype, normal individuals exhibiting the homozygous M phenotype. Twenty percent of Pi-type ZZ AATD infants present with a cholestatic type of neonatal hepatitis and develop a slowly progressive cirrhosis, and most die before reaching adulthood. Cirrhosis develops in about 10% of adult Pi ZZ homozygotes, coexistent emphysema occurring in most such individuals. In adults, primary hepatocellular and cholangiocellular carcinomas are frequently reported but are usually unsuspected during life. Specific globules resulting from the accumulation of a type of AAT are noted in the periportal liver cells of over 80% of homozygous and heterozygous Z AATD patients; liver disease is not always present. These globules are usually intracellular and increase in number and width with age. In infants, the percentage of liver cells containing globules is related to Pi-type and inversely related to the serum AAT concentration, but there is no relation to clinical state, liver cirrhosis, emphysema, or normal status. The defect in AATD resulting in the accumulation of these globules appears to reside in the liver. It is unlikely that they are directly hepatotoxic, although they may render the liver more susceptible to damage by other factors. An infectious factor has been suggested but none has been confirmed; an additional metabolic or nutritional factor has also been suggested. AATD infants have low birth weights, suggesting onset of liver disease in the fetus. The prognosis for patients with the neonatal hepatitis syndrome and Pi ZZ phenotype does not appear to be worse than for patients with the syndrome and normal serum AAT. Liver biopsies are of value in indicating or confirming AATD and in making an early diagnosis of hepatocellular carcinoma in AATD patients. Repeat liver biopsies are also of use in the follow-up of AATD patients.

6099 SURVIVAL OF PATIENTS WITH UNTREATED LIVER METASTASES FROM COLORECTAL CANCER. *(E.)* Baden, H.; Andersen, B. (Bispebjerg Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 16(1):221-223, 1975.

The effect of removal of the primary tumor on colorectal cancer patients with untreated liver metastases was studied. In October 1973, the survival status of 105 such patients, who had had the primary tumor removed between October 1962 and December 1972, was evaluated. The median survival time in this group was 10 months, nine patients being alive 108, 96, 93, 87, 56, 29, 18, 17, and 11 months after surgery. Among the nine patients still alive, the livers were definitely without metastases in two cases and their presence or absence was unconfirmed in the other seven. Of those who died, liver metastases were confirmed by autopsy in 77 cases and autopsies were not performed in the other 19 cases. These data, in conjunction with previous reports, indicate that the median survival time with untreated liver metastases is between 6 and 10 months after extirpation of the primary colorectal tumor; this value is considerably higher than would be expected in unselected series of liver metastases, suggesting that prognosis is better in patients who have had the primary tumor excised.

6100 ISOLATED LIVER NODULES. *(E.)* Shojania, N. G.; Hogg, G. R. (Univ. Manitoba, Med. Coll., Winnipeg, Canada). *Gastroenterology* 69(1): 28-34, 1975.

A series of seven cases of isolated liver nodules is reviewed. Two were encountered at surgery and five at autopsy. Autopsy incidence was slightly less than one in 1000 over a 6 yr period. In no case were there symptoms or signs relating to the nodule. Most cases had single nodules, but three cases had three separate nodules each. All 13 nodules were spherical, measured 0.5 to 9.5 cm in diameter, were pale to yellow in color and were unencapsulated. Light microscopy showed histological features resembling regenerating nodules in cirrhotic liver in 11 nodules; two nodules were fatty in makeup. Except for the nodular areas, the livers were all grossly and histologically normal. The nodules showed a visible scarcity of red cells, suggesting that the nodules were relatively ischemic. This may have resulted from abnormal blood flow. The nodules typically contained prominent vessels not associated with the portal tracts or central veins. They had abnormal, unevenly thickened walls composed mainly of fibrous tissue with a few sparse muscle fibers. The fatty changes in two of the nodules also supported ischemia as the etiological factor. It is suggested that the amount of fatty change parallels the degree of ischemia, or that the fatty nodules are the earliest ischemic effect, which may later undergo focal septal fibrosis and regenerative activity.

6101 HUMORAL AUTOIMMUNE PHENOMENA IN LIVER DISEASE. *(Ger.)* Berg, P. A. (Med. Clin., Univ. Tubingen, Germany). *Z. Gastroenterol.* 13(suppl.):257-263, 1975.

Of 105 patients with acute viral hepatitis, 78 were positive for hepatitis B antigen (HBAg). Immunofluorescence studies showed that, at onset, 80% had anti-smooth muscle antibodies, 35% had antibodies to vascular endothelium, and 12% had antinuclear antibodies; none of these patients had antimitochondrial antibodies. Antibodies to vascular endothelium occurred only in patients with blood group O. They were of the IgG type and reacted with an antigen related to blood group substance A. These antibodies occurred either in association with HBAg or, in some cases, appeared only after the patients became HBAg-negative. In contrast to anti-smooth muscle antibodies, antibodies to vascular endothelium persisted for some time after acute symptoms had subsided; they were absent in patients with autoimmune diseases. Antinuclear antibodies were present in low titers and, in most cases, reacted only with heterologous tissue. Both antinuclear antibodies and anti-smooth muscle antibodies were present in only 10-20% of HBAg-positive patients with chronic aggressive hepatitis. Antibodies to vascular endothelium were found in 30-40% of patients with blood group O, but were present in 50% of those with cryptogenic cirrhosis. Patients with autoimmune phenomena were defined as HBAg-negative and HB antibody-negative who also had high titers of antinuclear antibodies in the presence or absence of anti-smooth muscle and antimitochondrial antibodies. The simultaneous presence of antinuclear and anti-smooth muscle antibodies in titers above 1:40 suggests lupoid hepatitis with or without cirrhosis. Antimitochondrial antibodies occur in more than 95% of all patients with primary biliary cirrhosis, and the association of these antibodies with antinuclear antibodies indicates the simultaneous existence of an inflammatory, autoimmune process in both the bile ducts and hepatic parenchyma. A very rare form of chronic aggressive hepatitis, which occurs in only one case in 800, is associated with antimicrosomal antibodies. In addition to chronic aggressive hepatitis resulting from viral infection, drug-induced forms are found in which high antibody titers are present, but antibodies disappear after discontinuation of use of the drug.

6102 METABOLISM OF LITHOCHOLATE IN HEALTHY MAN. I. BIOTRANSFORMATION AND BILIARY EXCRETION OF INTRAVENOUSLY ADMINISTERED LITHOCHOLATE, LITHOCHOLYLGLYCINE, AND THEIR SULFATES. *(E.)* Cowen, A. E.; Korman, M. G.; Hofmann, A. F.; Cass, O. W. (Mayo Clin., Rochester, Minn.) *Gastroenterology* 69(1):59-66, 1975.

The metabolism of i.v. administered radiolabeled lithocholate, its glycine conjugate (lithocholylglycine), its sulfate (sulfothiocholate), and its sulfated glycine conjugate (sulfolithocholylglycine) in healthy subjects is reported. Lithocholate radioactivity was excreted rapidly, predominantly in bile. The excreted radioactivity had the chromatographic properties of glycine and taurine conjugates of lithocholate, of which 60% were sulfated. Lithocholylglycine also was excreted rapidly, predominantly in bile, and 60% of the excreted radioactivity was sulfated. Sulfolithocholate radioactivity was only partially conjugated (about 60%) in association with biliary excretion. Sulfolithocholylglycine was excreted unchanged in bile. Neither sulfated derivative showed appreciable excretion in urine, although both were excreted more slowly in bile than unsulfated free or conjugated lithocholate. The data suggest that unconjugated lithocholate, which is absorbed, is completely conjugated and partially sulfated before excretion, which occurs exclusively in bile. Since sulfation is not complete, some unsulfated lithocholate is always present in bile. It is concluded that this conjugated but unsulfated lithocholate, if reabsorbed, would be again partially sulfated during its next enterohepatic circulation.

6103 PARTIAL DEFICIENCY OF HEPATIC GLUCOSE-6-PHOSPHATASE IN AN ADULT PATIENT. *(E.)* Stamm, W. E.; Webb, D. I. (Univ. Washington Affil. Hosp., Seattle). *Arch. Intern. Med.* 135(8):1107-1109, 1975.

A case of partial hepatic glucose-6-phosphatase deficiency in a 40-yr-old woman is described. The manifestations in childhood were mild and unrecognized, consisting only of frequent epistaxis and bruising. Beginning at age 14, the patient suffered episodes of acute arthritis involving a variety of joints; except for persistent malaise and fatigue, the patient was asymptomatic between attacks. Consanguinity was not present; she had six normal siblings and bore three healthy children of her

own. She was of normal intelligence. Although blood glucose levels were low, actual hypoglycemia was not documented. The constellation of persistent acidosis, hyperlacticacidemia, hypercholesterolemia, hypertriglyceridemia, and hyperuricemia was repeatedly demonstrated over a period of several years. Unavailability of hepatic glucose-6-phosphate was demonstrated by a blunted hyperglycemic response to i.m. epinephrine and by a lack of blood sugar elevation following i.v. galactose. Other symptoms included bilateral arcus senilis and xanthelasma, s.c. tophi on the arms and legs, hepatic dullness, and decreased range of motion, synvovial thickening, s.c. nodules, and/or crepitus at the knees, elbows, ankles, wrists and small joints of the hands. A percutaneous liver biopsy demonstrated normal hepatocellular architecture with increased amounts of normally staining positive material. Partial glucose-6-phosphatase activity in this patient may explain her benign childhood course, emphasizing that patients with this deficiency may escape diagnosis during childhood, particularly if symptoms are mild or unrecognized. When acute gouty arthritis and tophaceous gout occur in an adolescent or young adult, the diagnosis of glucose-6-phosphatase deficiency should be considered.

6104 MORPHOLOGICAL CHANGES IN THE LIVER IN METABOLIC DISORDERS. *(Ger.)* Wohlgemuth, B. (Dep. Med., Karl Marx Univ., Leipzig, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(1):17-22, 1974.

Metabolic diseases affecting the liver result in deposition of material foreign to liver cells or in deposition of nonphysiological quantities of substances normally found there. Mild forms of toxic parenchymal damage are characterized by localized cellular necrosis, steatosis and jaundice in addition to changes in metabolic activation (anisocytosis and anisokaryosis, increase in the size of nucleoli, increase in the number of bi- and multinuclear hepatocytes, and changes in parenchymal structure). Kupffer cells are activated but show no signs of proliferation. In more severe damage the centrilobular areas are affected and necrosis of groups of cells, more marked steatosis, and leukocyte infiltration are observed. Cholestasis is occasionally present and is generally manifested by bile thrombi. The most severe form of parenchymal damage is dystrophy in which the entire liver becomes necrotic and there is no evidence of inflammation. The most common types of endogenous and exogenous metabolic diseases affecting the liver are briefly outlined.

6105 BILE ACIDS IN THE BLOOD AND URINE OF JAUNDICED AND NONJAUNDICED PATIENTS WITH VARIOUS LIVER DISEASES. *(Ger.)* Wildgrube, H. J.; Dittmann, K.; Erb, W. (Cent. Intern. Med., Univ. Frankfurt/Main, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:448-450, 1974.

Sulfate esters of bile acids were extracted from blood and urine specimens taken from 50 patients with compensated cirrhosis and 20 with hepatic metastases. The extracts were then fractionated on Sephadex LH 20, and sulfated bile acids were separated by gas-liquid chromatography of their trifluoro acetylated methyl esters on 3% QF-1. Patients with compensated cirrhosis had no evidence of jaundice (serum bilirubin levels lower than 1.5 mg/100 ml) or ascites, and none of the patients with hepatic metastases had primary tumors in the liver or biliary tract. The concentration of sulfate esters of blood bile acids in compensated cirrhosis ranged from 2 to 110 μM/l (mean 25.4 μM/l), while free and conjugated bile acid concentrations ranged form 7 to 130 μM/l (mean 31.8 μM/l). No significant correlation was found between these forms of bile acids. Most of the bile acids excreted in the urine of cirrhotics were in the form of sulfate esters. The rate of excretion averaged 0.84 μM/24 hr (maximum 2 μM/24 hr), and the content of free and conjugated bile acids was usually lower (around 0.68 μM/24 hr). These findings contrast with those observed previously in cirrhotics with epithelial or portal decompensation in whom both bile acid fractions were significantly increased. In both blood and urine, the concentration of cholic acid was significantly decreased. Patients with hepatic metastases had jaundice and increased concentrations of bile acids in their blood. However, the relative percentage and absolute concentrations of sulfated bile acids were completely independent of serum bilirubin concentrations. Many patients with high serum bilirubin concentrations (above 12 mg/100 ml) had relatively low sulfated bile acid concentrations, and these concentrations varied greatly during the day. These findings indicate that formation of sulfate esters of bile acids is not a protective mechanism occurring only in cholestasis.

6106 GRANULOMAS OF THE LIVER. *(E.)* Neville, E.; Piyasena, K. H. B.; James, D. G. (R. North. Hosp., London, England). *Postgrad. Med. J.* 51(596):361-365, 1975.

Clinical, radiological, and immunological data were reviewed in 138 cases of biliary hepatic granuloma, for factors helpful in defining etiology and prognosis, and for determining whether granulomatous hepatitis is a distinguishable condition. Of the patients, 75 had sarcoidosis, 26 had primary biliary cirrhosis, 23 had miscellaneous known disorders and 14 had idiopathic disease. Of the sarcoidosis group, 68% presented at 20-40 yr of age with no sex predilection, while 81% of the cirrhosis group presented at 30-50 yr and all but one were female. The miscellaneous group was predominately female, while the idiopathic group was mostly males over 50 yr of age. The sarcoidosis and cirrhosis groups presented with typical symptoms of the respective conditions. Liver function tests were most commonly abnormal in the cirrhosis and miscellaneous groups. Aspartate transaminase activity was most frequently abnormal (elevated in 85% overall), followed by alkaline phosphatase activity (62%). An abnormal chest radiograph was found in 55% of all patients, 84% of these in the sarcoidosis group. The Kveim-Siltzbach skin test was positive in 72% of the sarcoidosis group, but in no other group. Autoantibodies were most common in the cirrhosis group (79%). In 46 of the sarcoidosis

group followed up for 1 to 14 yr, 76% showed complete resolution, and 17% had chronic, persistent sarcoidosis. Of the cirrhosis group after a 1 to 10 yr follow-up, 30% died, 12% developed portal hypertension, and all had progressively abnormal liver function tests. Of the 14 idiopathic patients, five recovered spontaneously in 2 to 4 yr, and one recovered with anti-tuberculous chemotherapy. It is concluded that clinical picture, X-ray, blood, skin and microbiological tests result in diagnosis of cause in 90% of the cases; the term granulomatous hepatitis should be reserved for the undiagnosed 10% until follow-up reveals a more definitive cause.

6107 BENIGN HEPATOCELLULAR TUMOURS. *(E.)* Sorensen, T. I. A.; Baden, H. (Gentofte Hosp., Univ. Copenhagen, Denmark). *Scand. J. Gastroenterol.* 16(1):113-119, 1975.

The clinical, histological, and therapeutic experience in 250 cases of benign hepatocellular tumors is reviewed and an approach to the management of such tumors is suggested. Of all benign tumors and tumor-like lesions containing liver cells, only focal nodular hyperplasia (FNH), liver cell adenoma (LCA), and nodular liver cell adenoma are considered benign hepatocellular tumors. Of the 250 cases collected from Anglo-Saxon, German, French, and Scandinavian literature between 1923 and 1974, there were 141 cases of FNH, 69 cases of LCA (including 11 cases of nodular liver cell adenoma), and 40 unclassified tumors (UT). The frequency of these tumors may be increasing. In the clinical series, the female:male ratio was 3.7, with the incidence being highest among females in the fertile years and males between 0 and 30 yr; in the necropsy series, the female:male ratio was 1.6 and the median age was 50 yr. Most FNH were 4-7 cm, firm, and found in the left lobe, while most LCA were 8-15 cm, soft, and found in the right lobe. By light and electron microscopy, the cells and microstructures of the tumors resemble normal hepatic tissue. FNH is not potentially malignant, although LCA may be. Thirty-five percent of the FNH patients and 44% of the LCA patients presented with symptoms of moderate pain in the upper right abdominal quadrant, abdominal mass, and/or gastrointestinal upset. Complications, generally acute abdominal disease caused by hemorrhage and necrosis of the tumor, were the first manifestations in 38% of the LCA and none of the FNH patients. A hepatic tumor was preoperatively diagnosed by radiography, arteriography, and/or scintigraphy in 20% of the patients, and a preliminary diagnosis by biopsy was reported in 40 cases, with frozen section in 24, and with paraffin section in 16. Local resection or hemihepatectomy was used to remove the tumor entirely in 148 patients and partially in 34 patients. Total removal of the tumor generally cured the patient, and the outcome was good in 50% or more of the patients after partial removal. In patients with FNH, removal is not indicated if a major hepatic resection is required but is both effective and justified in the presence of complications or symptoms requiring treatment. In cases of LCA, the tumor should be removed. Enucleation of benign hepatocellular tumors is not advised and hormonal contraception should be discouraged in female patients. A 5-yr follow-up with ultrasonic scanning or scintigraphy is recommended.

6108 HEPATOSIS OF PREGNANCY. A CLINICAL STUDY OF 107 PATIENTS. *(E.)* Ylostalo, P.; Ylikorkala, O. (Dep. Obstet. Gynaecol., Univ. Oulu, Finland). *Ann. Chir. Gynaecol. Fenn.* 64(2):128-134, 1975.

The clinical experience in 107 patients with hepatosis of pregnancy is reported and compared with that in 61 controls with normal pregnancies. All cases were evaluated with regard to the date and duration of delivery, birth weight and condition of the neonate, and weight of the placenta. The hepatosis group was also given liver function tests and a glucose tolerance test, and the urinary estrogen levels were measured daily. The duration of the first stage of delivery was slightly ($p < 0.05$) shortened in the hepatosis group, and delivery took place more than 2 weeks earlier in this group than in the controls. There were two fetal deaths in the hepatosis series. The average Apgar scores at 1 and 15 min were slightly but significantly lower in the hepatosis group than in the controls, with no differences in scores according to sex. In addition, the average birth weight was slightly lower in the hepatosis group, and 11.9% of the infants in this group weighed less than 2.5 kg at birth. The absolute and relative placental weights did not differ between the two groups, although there were histologically evident maturing defects in the placenta in 20 of the 58 hepatosis cases studied. The liver function tests confirmed the cholestatic nature of the disease, yielding elevated aminotransferase, alkaline phosphatase, and bilirubin levels. The thymol turbidity test was within normal limits, indicating that the hepatosis series may have included cases of hepatitis. Neither glucose nor daily urinary estrogen levels differed significantly from normal. Fifty-five percent of the multiparous hepatosis patients had suffered from this disorder during earlier pregnancies. The fetal survival rate in hepatosis of pregnancy can be considerably improved by intensive hospital treatment during pregnancy.

6109 ON THE EXISTENCE OF INTRAHEPATIC BILE DUCT SHUNTS. *(E.)* Aiginger, P.; Polterauer, P.; Tizian, C. H. (Univ. Vienna, Med. Sch., Austria). *Anat. Embryol.* 147(1):111-116, 1975.

A combined scintigraphic-radiological method was used to study the existence of intrahepatic shunts between the bile ducts of the right and left duct systems in 28 human autopsy specimens. The average age at death was 39 yr and there were equal numbers of males and females. The proximal section of the common bile duct was exposed, and meglumine diatrizoate and Tc-99m Pertecnetat were injected in different orders into the left or right intrahepatic bile duct system after application of a water-tight ligature immediately proximal to the hepatic bifurcation. In all cases, passage of the injected substances into the periphery was observed. Although very small biliary capillaries were visualized, the substances did not appear to en-

ter the contralateral bile duct system. Similarly, intralobular shunts were not observed following bilateral drainage of the caudate lobe. Despite the great difference in viscosity in the substances used, identical results were obtained with the combined radiologic-scintigraphic method. On the basis of these results, no conclusions regarding function can be made.

6110 REYE SYNDROME: A CORRELATED ELECTRON-MICROSCOPIC, VIRAL, AND BIOCHEMICAL OBSERVATION. *(E.)* Tang, T. T.; Siegesmund, K. A.; Sedmak, G. V.; Casper, J. T.; Varma, R. R.; McCreadie, S. R. (Milwaukee Child. Hosp., Wis.). *JAMA* 232(13): 1339-1346, 1975.

Liver biopsy specimens from two patients with Reye syndrome were examined ultrastructurally and for viral particles and urea-cycle enzyme activity. The patients, 10- and 14-yr-old boys, were hospitalized with symptoms of upper respiratory tract infection, persistent vomiting, delerium, combative behavior, and hyperammonemia. The first patient died 15 hr after admission and autopsy speciments of lung, liver, and heart were obtained. The second patient recovered and was discharged after nine days; a liver biopsy specimen was obtained on the second day after hospitalization. In addition, urine, serum, and throat swab samples were obtained from both patients. The liver specimens were examined by electron microscope and attempts were made to isolate viral particles from the other tissues and urine and throat specimens after inoculation into cultures of human foreskin fibroblasts, WI-38, HeLa, HEp-2, and grivet monkey kidney cells. The serum samples were tested for antibodies to various viruses by microcomplement fixation and hemagglutination inhibition procedures and were assayed for hepatic ornithine transcarbamoylase, hepatic carbamoyl phosphate synthetase, arginase, and glycogen. The hepatocytes from both patients contained a large amount of glycogen and degenerating mitochondria. Numerous herpes-like virus and myxovirus/paramyxovirus particles were seen in the spaces of Disse and sinusoids, and there was a leakage of glycogen granules into these areas. Cytomegalovirus was isolated from the heart and liver speciments of the first patient, but no particles were isolated from the liver specimen of the second patient or urine specimens of either patient. The results were supported by the serologic tests, and serum hepatic ornithine transcarbamyolase levels in both patients were subnormal. The pathogenesis in Reye syndrome therefore appears to involve an initial synergistic insult to the liver by mixed types of viruses and subsequent breakdown of the urea cycle, Krebs cycle, and possibly other hepatic functions.

6111 FATTY METAMORPHOSIS IN PATIENTS WITH JEJUNOILEAL BYPASS. *(E.)* Salmon, P. A.; Reedyk, L. (Dep. Surg., Univ. Alberta, Edmonton, Canada). *Surg. Gynecol. Obstet.* 141(1):75-84, 1975.

The natural course of fatty metamorphosis of the liver was studied in 106 patients who underwent end-on-jejunoileal bypass between 1967 and 1970. Wedge or percutaneous needle liver biopsies were performed on all patients at the time of surgery and at various intervals thereafter. The biopsy material was examined for evidence of fatty metamorphosis and cirrhosis, and liver function tests were performed within a few hr of each biopsy. Two-thirds of all preoperative patients had some degree of fatty metamorphosis of the liver and at least 8-10% had marked steatosis prior to the bypass procedure. Marked fatty metamorphosis was most common in the massively obese patients, while the incidence of moderate to minimal fatty infiltration was highest in the less obese patients; there was, however, no strict correlation between degree of fatty infiltration and obesity. After surgery, 95% of the patients showed moderate to severe fatty metamorphosis within the first 300 days. Subsequently, the incidence of fatty infiltration rapidly decreased, so that by 1500-2000 days postsurgery, the incidence of severe steatosis had returned to preoperative levels; only those patients with marked postoperative involvement and complications late in the postoperative course had a severe degree of fatty metamorphosis after 1000 days. The liver function tests were of no value in detecting quiescent cirrhosis or minimal to moderate degrees of fatty metamorphosis, and showed only inconsistent changes in those patients with severe steatosis. Cirrhosis occurred in one patient after the liver was completely cleared of fat and was present to a degree in another patient at the time of bypass. Mononuclear cell infiltration of the portal tracts was found in about 50% of the preoperative biopsies, but did not correlate well with the incidence of hepatic steatosis. Because severe postoperative fatty infiltration in patients with preoperatively normal or minimally infiltrated livers occurred during the period of rapid weight loss, it is suggested that the extensive mobilization of depot stores of fat to fulfill the caloric requirements of the patient results in the accumulation of triglyceride in the liver. Steatosis may be further aggravated by the reduction in protein absorption and concomitant reduction in the formation of low-density lipoproteins in the liver.

6112 LIVER FAILURE IN THE POSTOPERATIVE PATIENT: THE ROLE OF SEPSIS AND IMMUNOLOGIC DEFICIENCY. *(E.)* Norton, L.; Moore, G.; Eiseman, B. (Univ. Colorado Med. Cent., Denver). *Surgery* 78(1): 6-13, 1975.

The cellular immune response was studied in 20 of 73 patients who developed liver failure and coma preoperatively. Quantitation of the cellular immune response in these patients, most of whom were alcoholic, included a differential leukocyte count, reduction of nitroblue tetrazolium by the blood neutrophils, the lymphocyte phytohemagglutinin (PHA) response, the ratio of T:B lymphocytes, serum immunoglobulin (Ig) A, G, and M levels, serum zinc concentration, lymphocyte sensitization to a skin test antigen, and skin reactions to intradermal tuberculin, mumps, and *Candida albicans* antigen. Among the 73 original patients, bacterial sepsis, generally of i.p. origin, was a major contributing factor in precipitating hepatic failure in 27; the mortality rate in this group was 48%. Massive blood loss and prolonged hypotension precipitated coma in 14 pa-

tients, of whom 10 died, bleeding from esophageal varices precipitated liver failure in 25 patients, 23 of whom died. Four patients had serum hepatitis and two died, and liver failure was attributed to the toxic effects of anesthesia in three patients, two of whom died. The cellular immune response in the 20 cirrhotic patients showed a reduced leukocyte count, a significant reduction in the lymphocyte PHA response, T lymphocyte count, and serum zinc concentration, and significant increases in serum IgA, IgG, and IgM. The percentage of cirrhotic patients reacting positively to intradermal tuberculin, mumps, and *Candida albicans* antigen was also increased over control levels. The results suggest the presence of a depressed cellular immune response in cirrhotic patients; thus, once such patients become septic, they are at double risk since they cannot mount a proper immunologic response. An aggressive attitude toward the elimination of infection is justified, therefore, as is re-exploration in any postoperative or postinjury patient who has any possibility of having undrained i.p. sepsis.

6113 SIGNIFICANCE OF ELEVATED LIVER ALKALINE PHOSPHATASE IN SERUM. *(E.)* Brensilver, H. L.; Kaplan, M. M. (New England Med. Cent. Hosp., Boston, Mass.). *Gastroenterology* 68(6):1556-1562, 1975.

To verify the hepatic origin of the band L isoenzyme, polyacrylamide gel electrophoresis was used to fractionate the serum alkaline phosphatase in 317 patients in whom the cause of the alkaline phosphatase elevation was not apparent or in whom the elevation appeared out of proportion to the extent of the disease present. In 87 patients with increased L band activity who did not have clinically apparent liver disease, liver biopsies and liver function tests were also performed. Of the original 317 sera, the alkaline phosphatase elevation was due to band L in 253. These 253 patients included 166 with hepatic disorders generally associated with such an elevation; the mean alkaline phosphatase in this group was 19.6 Bodansky U, compared with 8.8 in the 87 patients with increased L band activity in whom there was no apparent liver disease. In 19 of the latter 87 cases, liver disease was further excluded by liver biopsy, liver function tests, and/or laparotomy. The 87 cases also included 19 with acute coronary vascular disease or congestive heart failure in whom unrecognized passive congestion of the liver may have contributed to the alkaline phosphatase elevation. None of the other patients, including 24 with Hodgkin's disease and nine with tumors, had recognizable liver disease. In light of these findings, studies were undertaken to confirm the hepatic origin of band L. Band L and alkaline phosphatase extracted from human liver migrated together on polyacrylamide gels before and after digestion with *Vibrio cholerae* neuraminidase and they had identical pH optima, sedimentation coefficients, Michaelis constants, and rates of inactivation at 55.5 C. However, they had different rates of inactivation in 3 M urea. Overall, the data indicate that band L is of liver origin and that elevation of the hepatic alkaline phosphatase isoenzyme may be a nonspecific finding in certain patients.

6114 THE RELATIONSHIP OF RED CELL MEMBRANE LIPID CONTENT TO RED CELL MORPHOLOGY AND SURVIVAL IN PATIENTS WITH LIVER DISEASE. *(E.)* Powell, L. W.; Halliday, J. W.; Knowles, B. R. (R. Brisbane Hosp., Queensland, Australia). *Aust. N. Z. J. Med.* 5(2): 101-107, 1975.

The relation between RBC membrane lipid content and RBC morphology and survival was studied in 15 patients with hepatic cirrhosis and seven patients with cholestatic jaundice. RBC morphology was also studied in 56 additional patients with hepatic cirrhosis to determine the prevalence of deformed RBC in this disease. Serum lecithin:cholesterol acyltransferase (LCAT) activity and RBC lipids were determined in the original 22 cirrhotic and jaundiced patients and in seven healthy volunteers. RBC morphology was studied in all liver disease patients using air dried smears, wet preparations, and scanning electron microscopy of the peripheral blood. RBC survival was measured by the radioactive chromium technique. Both RBC cholesterol and RBC phospholipid were significantly elevated over control values in the patients with liver disease, the type of disease having no significant correlation with the degree of elevation. In two patients with significant numbers of "spur" poikilocytes in the peripheral blood, RBC cholesterol was significantly increased but RBC phospholipid content was normal. The serum cholesterol levels did not correlate significantly with the RBC lipid levels. Serum LCAT activity was reduced in liver disease, the level of enzyme activity correlating significantly with the serum total bilirubin; it did not correlate significantly with RBC cholesterol or phospholipid values or serum cholesterol content. Peripheral blood smears from the liver disease patients regularly showed macrocytes, target cells, and some poikilocytes, but the number of deformed RBC did not correlate with RBC lipid content or RBC survival. Of the additional 56 hepatic cirrhosis patients in whom morphology was studied, distorted RBC were found in only two. RBC survival was reduced in most patients, but the degree of hemolysis did not correlate significantly with RBC lipid, the presence or degree of splenomegaly, or serum iron, folate, or vitamin B_{12} concentrations. Marked hemolysis was, however, correlated with a large number of spur poikilocytes. The results indicate that only in patients with spur-cell anemia do the morphologic alterations lead to the premature removal of cells from the circulation.

6115 HEPATOCELLULAR CARCINOMA AND A VARIANT ALKALINE PHOSPHATASE. *(E.)* Higashino, K.; Ohtani, R.; Kudo, S.; Hashinotsume, M.; Hada, T.; Kang, K. Y.; Ohkochi, T.; Takahashi, Y.; Yamamura, Y. (Osaka Univ. Sch. Med., Japan). *Ann. Intern. Med.* 83(1):74-78, 1975.

The clinical, laboratory, and pathologic findings in patients with a variant alkaline phosphatase are reported, and the usefulness of this enzyme in the diagnosis of hepatocellular carcinoma is discussed. Serum samples were obtained from 100 control subjects, 13 patients with primary hepatoma, and 117 patients with other liver and extrahepatic diseases; liver and tumor tissues were obtained at autopsy from 13 patients with hepatoma and 94 patients with other

diseases. Alkaline phosphatase was extracted from these tissues, fractionated, and subjected to polyacrylamide gel electrophoresis. Screening for serum alpha-fetoprotein was done by a micro double diffusion method. Four of the 13 primary hepatoma patients had the variant serum alkaline phosphatase, which resembles the placental D-variant but differs from it in electrophoretic mobility, pH optimum, heat stability, and inhibition by phosphate. The appearance of the variant enzyme was independent of serum alkaline phosphatase activity and was not found in the sera of patients with diseases other than hepatocellular carcinoma. Alpha-fetoprotein was found in the sera of 10 of the 13 primary hepatoma patients, the occurrence of the deviant serum enzyme being independent of the alpha-fetoprotein concentration and the appearance of the Regan or Nagao isoenzymes. The tumor tissues of three patients with the variant serum enzyme were examined and all were found to contain the enzyme in question. Since all of the patients in whom the variant alkaline phosphatase was present had a massive type of hepatocellular carcinoma with grade III differentiation by Edmondson's classification, the detection of this enzyme in the serum may be of value in confirming a diagnosis of hepatoma.

6116 LIVER MORPHOLOGY AND LIVER FUNCTION BEFORE AND AFTER INTESTINAL SHUNT OPERATION FOR OBESITY. *(E.)* Dano, P.; Nielsen, O. V.; Petri, M.; Jorgensen, B. (Rigshosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 10(4):409-416, 1975.

The influence of obesity and intestinal shunt operation on liver function and morphology was studied in 49 patients. Liver biopsy and liver function tests were performed in 17 patients before and 14 months after the bypass operation. In another 20 patients, liver biopsy was performed postoperatively only, and in 13 patients preoperatively only. Three types of operations were performed: in type I, 36 cm of proximal jejunum was anastomosed end-to-side to 12 cm of terminal ileum; in type II, 24 cm of proximal jejunum was anastomosed to 24 cm of terminal ileum; and in type III, 12 cm of proximal jejunum was anastomosed to 36 cm of terminal ileum. Weight loss after the operation was most pronounced during the first 6 months and the degree of loss did not vary significantly according to the type of operation. In 29 patients with liver biopsy before surgery, steatosis was present in 25. In the 17 patients examined 14 months after surgery, the degree of hepatic steatosis was increased in seven, decreased in eight, and unchanged in two. In eight patients, a further biopsy 15 months later showed decreased steatosis in five and no alteration in three. There was no correlation between the rate of weight loss in the first 12 months and the change in hepatic steatosis. In the 20 patients examined postoperatively only, all had hepatic steatosis; in 10 patients examined several months later, hepatic steatosis was decreased in five and unchanged in five. No patient developed cirrhosis during the time of observation, although one patient each in the preoperative and postoperative only groups had cirrhosis. Within the first postoperative yr, there was a significant increase in the serum alanine-aminotransferase levels and a minor increase in alkaline phosphatases and BSP; these changes did not correlate with the rate of weight loss or degree of steatosis. The incidence of gallbladder disease was 15% preoperatively and two additional patients were cholecystectomized postoperatively. The results indicate that both serial biopsies and quantitative measurements of hepatic lipid are necessary when further information on the influence of the bypass operation on the liver is desired. The intestinal shunt operation is still recommended in selected cases of obesity.

6117 α-FETOPROTEIN IN NONNEOPLASTIC HEPATIC DISORDERS. *(E.)* Bloomer, J. R.; Waldmann, T. A.; McIntire, K. R.; Klatskin, G. (Yale Univ. Sch. Med., New Haven, Conn.) *JAMA* 233(1):38-41, 1975.

To define the specificity of the radioimmunoassay for serum α-fetoprotein as a diagnostic test for hepatoma, serum α-fetoprotein levels were measured by radioimmunoassay in 473 patients with biopsy-proven nonneoplastic disorders. The patients ranged in age from 3 yr to 80 yr; their diseases included viral hepatitis, inactive postnecrotic cirrhosis, chronic active hepatitis, drug-induced hepatitis, primary biliary cirrhosis, alcoholic liver disease, extrahepatic biliary disease, obstructive jaundice, and other miscellaneous hepatic disorders. α-Fetoprotein levels were also measured in 75 patients with proved hepatocellular carcinoma, 350 patients with nonhepatic nonneoplastic disease, and 130 healthy volunteers. Among the patients with nonneoplastic hepatic disorders, 22% had α-fetoprotein values greater than 40 ng/ml, levels exceeding 40 ng/ml being found in 30% of the patients with various types of hepatitis, and in 0-15% of those with other diseases. None of the healthy controls and only one of the patients with nonhepatic benign diseases had an α-fetoprotein value over 40 ng/ml. Among the 75 hepatoma patients, serum α-fetoprotein levels exceeded 40 ng/ml in 69% and exceeded 3000 ng/ml in 48%. The results indicate that radioimmunoassay may, because of its sensitivity, become the method of choice for measuring α-fetoprotein levels in patients with primary hepatoma, although some specificity for the diagnosis of hepatoma will be lost. The occurrence of elevated serum α-fetoprotein levels in several other nonneoplastic hepatic disorders may reflect hepatic regeneration.

6118 DIAGNOSTIC VALUE OF SERUM BILE ACIDS. *(Ger.)* Schwarz, H. P.; Paumgartner, G.; Preisig, R. (Inst. Clin. Pharmacol., Univ. Bern, Switzerland). *Schweiz. Med. Wochenschr.* 105(17): 533-535, 1975.

Fasting serum bile acids, serum bilirubin, SGOT, alkaline phosphatase, the galactose excretion test, and BSP retention and clearance tests were performed on 60 normal controls, 14 patients with Gilbert's syndrome, four with Dubin-Johnson syndrome, and 48 with a variety of liver diseases. Bile acids were extracted with Amberlite XAD-2, incubated with 3α-hydroxysteroid dehydrogenase, and measured fluorimetrically. In controls, serum bile acid levels

ranged from 1.6 to 9.2 μM/liter (means 6.1 ± 1.7 μM/liter for males and 5.9 ± 1.7 μM/liter for females). All 14 patients with Gilbert's syndrome and two of the four with Dubin-Johnson syndrome had values within the normal range; the two other patients with Dubin-Johnson had slightly elevated levels (10.5 and 10.6 μM/liter, resp.). With the exception of two patients with fatty liver and one with cryptogenic cirrhosis, patients with all other liver diseases (94%) had increased serum bile acid levels. However, a differential diagnosis could not be established on the extent of this increase. BSP retention gave abnormal results in 96%, BSP clearance in 90%, SGOT in 88%, galactose excretion in 65%, alkaline phosphatase in 60%, and serum bilirubin in 40%. Significant correlations were found between serum bile acid levels and BSP retention, the BSP clearance constant k_1, and serum total bilirubin values. However, large discrepancies were noted in some cases: 27 of the 45 patients with increased bile acid values had normal serum bilirubin levels. When serum bile acid determinations were repeated 2 hr after a standard breakfast (egg, bread and butter, coffee), significant postprandial increases occurred in all eight patients with cirrhosis and all 10 controls tested. However, bile acid concentrations of more than 10 μM/liter were never observed in controls. This relatively simple method for determining serum bile acids appears to be a very sensitive method for diagnosing liver disease. In patients with normal or borderline values, determinations should be repeated 2 hr after a standard breakfast. Since serum bile acid values in three patients with cirrhosis increased significantly after portacaval shunt surgery and excretory liver function, measured with the BSP transport maximum, remained the same, the increased bile acid concentrations found in patients with liver disease appear to be due both to impaired liver cell function and porto-systemic shunts.

6119 CRITERIA FOR THE ACTIVITY OF PATHOLOGICAL CONNECTIVE TISSUE PROLIFERATION IN CHRONIC LIVER DISEASE. *(Ger.)* Grasedyck, K.; Helle, M.; Lindner, J.; Langness, V. (1st Med. Clin., Univ. Hamburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80: 503-506, 1974.

Total collagen contents, expressed as hydroxyproline, and collagen synthesis, using prolyl hydroxylase activity as the criterion, were measured in the livers of 10 cadavers. Collagen was also separated into four fractions on the basis of solubility in neutral salt and acid solutions. The collagen content increased proportionally with the extent of fibrosis or cirrhosis, but no relation was observed between the extent of fibrosis and the distribution pattern for individual collagen fractions. However, total collagen values obtained on fat-free dry tissue agreed well with values for the collagen fractions. Measurements of collagen in serial sections from cadaver liver showed that scattering amounted to 10-20%. With the technique employed, it is possible to determine total collagen in tissue samples as small as 20-30 mg wet weight or 5-8 mg dry weight. Hydroxyproline levels were 10-20 μg in normal livers and more than 100 μg in pathological livers. Preliminary studies demonstrated that the use of prolyl hydroxylase activity as a criterion for collagen synthesis gave significantly different values for patients with cirrhosis and those with normal livers. Although these findings give no information on correlations between the extent of newly formed connective tissue or the prognosis, insight into fibrosis can be obtained if the total collagen content and prolyl hydroxylase activity are determined in needle biopsies.

6120 CYTOCHROME P-450-DEPENDENT 6α-HYDROXYLATION OF TAUROLITHOCHOLIC ACID IN MAN. *(Ger.)* Czygan, P.; Greim, H.; Trultsch, D. (Med. Clin., Univ. Heidelberg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:445-447, 1974.

To demonstrate that cytochrome P450 takes part in 6α-hydroxylation of taurolithocholic acid in normal human liver microsomes, the reaction kinetics, CO inhibition, and the reversibility of CO inhibition by light at 450 nm were investigated. The 6α-hydroxylation of taurolithocholic acid followed Michaelis-Menten kinetics up to a substrate concentration of approximately 0.3 mM. Inhibition occurred at higher concentrations due to the detergent action of taurolithocholic acid. The K_M was 0.21 mM, a value that agrees with those found in other cytochrome P450-dependent reactions, and the maximum reaction rate was 0.79 nM/min/mg microsomal protein. When this reaction was carried out in the presence of 2-48% CO and 4% O_2 for five min at 30 C, inhibition of 6α-hydroxylation increased with increasing CO concentrations. A Warburg distribution constant, K, of 1.8 was obtained at 50% inhibition, a value similar to those reported for other cytochrome P450-dependent reactions. CO-induced inhibition of 6α-hydroxylation was reversed most rapidly when the incubation mixture was exposed to monochromatic light having a wavelength of 450 nm. These findings can be regarded as definite proof that cytochrome P450 takes part in 6α-hydroxylation of taurolithocholic acid. This may be the first demonstrated cytochrome P450-dependent reaction in bile acid metabolism in man. This reaction might be useful in explaining the etiology of intrahepatic cholestasis since drugs which cause this condition and are metabolized by the cytochrome P450-enzyme system would interfere with hydroxylation of taurolithocholic acid. However, it is not known whether this hepatotoxic bile acid would contribute to intrahepatic cholestasis or whether it would only result in potentiation.

6121 HISTOGENETIC STAGES BETWEEN POLYCYSTIC LIVER AND "CONGENITAL HEPATIC FIBROSIS". STUDY OF TWO CASES IN ADULTS. *(It.)* Tomasino, R. M.; Genova, G.; Messina, L. (Inst. Anat. Pathol. Histol., Univ. Palermo, Italy). *Arch. De Vecchi Anat. Patol.* 59(2):261-292, 1973.

Case reports are presented for a 39-yr-old woman with polycystic liver associated with polycystic kidney and a 40-yr-old woman with "congenital fibrosis of the liver." From histological findings it is concluded that these two conditions are actually early and late forms, resp., of the same

disease. Laparoscopy of the first patient revealed many cystic protuberances of a greyish-white color, which varied in size from a hazel nut to a hen's egg. A needle biopsy showed dilatation and congestion of the sinusoids; their lumens were filled with many blood cells. Inflammatory infiltrates were present in the periportal region and in the parenchyma; these were associated with diffuse fatty liver and signs of regeneration. The diameters of dilated ductal formations, found in the parenchyma and areas distant from the portal triad, were consistent with those of intralobular bile ducts; these were covered with cylindrical epithelium in which the nuclei were shifted toward the base of the cell and the cytoplasm stained with periodic acid-Schiff's reagent. These structures, which occurred singly or in groups of two or three, consisted only of epithelial covering resting on a very thin fibrous wall surrounded by a layer of hepatocytes; small quantities of bile were present in their cavities. The second patient had a five-yr history of splenomegaly associated with gastrointestinal discomfort. At laparoscopy the spleen and liver were increased in size; the liver was hard, greyish, and "precirrhotic" in appearance. Histological examination of a large fragment of the liver showed thick bands of scarring which separated the parenchyma and surrounded roundish islands of the muralium simplex. In some areas hepatocytes contained lipofuscin pigment. Proliferation of thick bands of connective tissue surrounding the bile ducts was found in all areas examined. As in the first case, proliferation of the intralobular bile ducts was detected; it was not associated with either inflammatory changes or parenchymal necrosis.

6122 A CASE OF ENCAPSULATING PERIHEPATITIS TREATED BY PORTACAVAL ANASTOMOSIS AND HEPATIC DECORTICATION. *(Fr.)* Chevrel, J. P.; Gueraud, J. P. (Franco-Musulman Hosp., Bobigny, France). *Chirurgie* 100(11):806-815, 1974.

A 44-yr-old woman, hospitalized for recurrent hematemesis, had alcoholic cirrhosis associated with portal hypertension and esophageal varices. She had a history of tuberculosis and had undergone nephrectomy on the left 11 yr previously. Clinical examination showed hepatosplenomegaly, conjunctival subicterus, ascites, and Cruveilhier-Baumgarten syndrome. When the peritoneum was opened for omphalography, the liver did not appear nodular and cirrhotic, but resembled the icing of a cake. Although the patient later stopped drinking and gained weight, her liver became more enlarged and she was readmitted to undergo hepatic decortication in association with an end-to-side portacaval shunt. A biopsy of the liver taken at this time confirmed the existence of micronodular cirrhosis associated with moderate steatosis and Curschmann's encapsulating perihepatitis. The patient's recovery was uneventful and one year after surgery her condition has stabilized: the liver is normal in size, liver function tests have improved considerably, and there has been no recurrence of hematemesis, ascites, or edema of the lower extremities; portacaval encephalopathy has not developed, but the patient is currently suffering from polyneuritis.

6123 THE ISOLATION AND CHARACTERIZATION OF PLASMA LIPASE OF HEPATIC ORIGIN: PRELIMINARY CLINICAL FINDINGS IN PATIENTS WITH VARIOUS LIVER DISEASES. *(Ger.)* Klose, G.; Greten, H. (Med. Clin., Heidelberg Univ., Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:1272-1274, 1974.

The activities of a lipase of hepatic origin, which hydrolyzes triglycerides, and of a lipoprotein lipase from fatty tissue were determined in heparinized blood taken from 13 patients with a variety of liver diseases (acute and chronic hepatitis, fatty liver and cirrhosis) and from eight normal controls. Plasma was extracted successively with acetone, heptane, ethanol-ether, and ether at 4 C to remove lipids which normally form a complex with heparin. Proteins were dissolved in barbital buffer (pH 7.4) and incubated with ^{14}C-labeled triolein to which albumin and detergents had been added for 30 min at 27 C. The ^{14}C-labeled free fatty acids were then isolated by passage through an ion-exchange resin and their activity was determined. More than 50 μM/5 ml/hr of free fatty acids were found in total postheparin plasma from seven of the eight controls, while less than 40 μM/5 ml/hr were found in serum from 12 of the 13 patients with liver disease. Calculation of the ratios between triglyceride lipase of hepatic origin and lipoprotein lipase from fatty tissue showed values of less than 2:1 for most of the patients and values as high as 10:1 in the control group. It is suggested that increased triglyceride concentrations in the blood of patients with liver disease are due, at least in part, to a decrease in plasma triglyceride lipase synthesized in the liver.

6124 BILE ACIDS IN THE LIVER AND SERUM IN VARIOUS LIVER DISEASES. *(Ger.)* Greim, H.; Czygan, P. (Toxicol. Inst., Univ. Tubingen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:443-445, 1974.

So that comparisons could be made between serum and liver bile acids, a technique was developed for determining individual bile acids in needle liver biopsies weighing 5 to 15 mg. Liver was solubilized in 3.4 N KOH which was then neutralized with HCl. Glycine and taurine conjugates were hydrolyzed with 50 μl of cholylglycyl hydrolase in a solution containing 3.5 ml of buffer mixture at pH 5.6 (0.1 M acetate buffer and 0.01 M each of β-mercaptoethanol and EDTA-Na_2). After ether extraction, thin-layer chromatography with a hexane-ethyl acetate (9:1) eluant, and methylation, bile acids were determined by gas-liquid chromatography as their trifluoroacetyl or trimethylsilyl derivatives. Serum bile acids were determined by hydrolyzing 0.5 ml of serum with 3 ml of buffer mixture and enzyme and proceeding as above. Cholic, deoxycholic and chenodeoxycholic acid contents of the liver were compared with their serum concentrations in three patients with liver disease (chronic aggressive hepatitis, sclerosing cholangitis, and cholestasis caused by testosterone) and in six patients with no evidence of liver disease. The ratio of serum to liver bile acid varied between 1:2 and 1:30 so that serum bile acid concentrations give no information about their content in the liver. In part, this can be attributed to the method used which

also detects extracellular bile acids. However, with the exception of the patient with chronic aggressive hepatitis, the distribution pattern of bile acids was almost identical in liver and serum. This finding was confirmed in three patients with primary biliary cirrhosis and increased cholic acid levels and in three patients with chronic persistent hepatitis and relatively small increases in bile acid concentrations in both compartments. Therefore, determination of individual bile acids in the serum does give information about the bile acid distribution pattern in the liver.

6125 HEPATIC RETICULOENDOTHELIAL HOST DEFENSE FAILURE FOLLOWING SURGICAL TRAUMA. *(E.)* Scovill, W. A.; Saba, T. M. (Albany Med. Coll., N.Y.). *Arch. Surg.* 110(8):954-959, 1975.

The potential pituitary adrenal influences on postoperative reticuloendothelial function was evaluated in male dogs pretreated with dexamethasone sodium phosphate (3 mg i.m. twice a day for 3 days, with the last dose increased to 6 mg). The operative procedure consisted of right hemicolectomy. The reticuloendothelial function was evaluated by a colloid clearance technique and the plasma opsonic activity was determined before and after surgical trauma by an *in vitro* bioassay. Three hr after surgery significant phagocytic depression was evident in untreated animals by an almost 50% decrease in the global phagocytic index K (vascular clearance of the i.v. injected colloid). There was a 30% decline in opsonic activity. Both the global phagocytic index K and the plasma opsonic activity of control, dexamethasone-pretreated animals were not significantly different than control nontreated animals. In dexamethasone-pretreated dogs, the K value of 0.79 was not significantly changed from control nontraumatized animals. Although opsonic activity declined in the dexamethasone-treated animals, values were not significantly different from control. Cortisone acetate or ACTH over a wide dosage range manifested no depressing effect on *in vitro* phagocytosis. The manipulation of pituitary adrenal function appears to have a substantial postoperative influence on hepatic macrophage function. The precise mechanism of this regulatory influence is unknown but it would appear to be of a hemestatic or permissive nature at the cellular level.

6126 QUANTITATIVE DETERMINATION OF THE ABNORMAL LIPOPROTEIN OF CHOLESTASIS, LP-X, IN LIVER DISEASE. *(E.)* Ritland, S. (Univ. Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 15(11):5-15, 1975.

Quantitative determination of an abnormal lipoprotein, lipoprotein-X (LP-X) was performed in 15 patients with acute hepatitis, seven with toxic hepatitis, 10 with primary biliary cirrhosis, one with other cirrhosis, two with cholestasis of pregnancy, 10 with sclerosing cholangitis, six with cholecocholithiasis, 30 with extrahepatic biliary obstruction by tumors, and 17 patients with an uncertain diagnosis. Blood samples were drawn in the fasting state, and determinations of LP-X, free and total cholesterol, and α-lipoprotein, serum bilirubin, alkaline phosphatases, albumin, and SGOT were made. Great variations in the plasma LP-X concentration were demonstrated in the 45 patients with intrahepatic cholestasis and in the 36 patients with extrahepatic cholestasis, although the median LP-X concentrations were significantly higher in the former group (158 mg/100 ml) than in the latter group (25 mg/100 ml). However, the highest LP-X level in the intrahepatic cholestasis group (252 mg/100 ml) was exceeded by 42% of the extrahepatic biliary obstruction patients, 33% of whom had plasma LP-X levels greater than 400 mg/100 ml. The plasma LP-X concentration was significantly correlated with free cholesterol, plasma alkaline phosphatases, and serum bilirubin levels, but seemed superior to the latter two parameters in differentiating between intrahepatic and extrahepatic cholestasis. Plasma LP-X concentrations exceeding 400 mg/100 ml are highly indicative of extrahepatic biliary obstruction.

6127 STEROID HORMONES AND THEIR BINDING IN PLASMA OF MALE PATIENTS WITH FATTY LIVER, CHRONIC HEPATITIS AND LIVER CIRRHOSIS. *(E.)* Kley, H. K.; Nieschlag, E.; Wiegelmann, W.; Solbach, H. G.; Kruskemper, H. L. (Med. Univ. Clin., Dusseldorf, Germany). *Acta Endrocrinol. (Kbh.)* 79(2):275-285, 1975.

Plasma estrone (E_1), estradiol (E_2), testosterone (T), cortisol (F), and luteinizing hormone (LH) levels were studied in 10 male patients with fatty liver, six male patients with chronic hepatitis, 15 male patients with cirrhosis of the liver, 25 healthy young (19-40 yr) male subjects, and 25 healthy older males (61-89 yr); the percentage of bound T, E_1, E_2, and F in the plasma was also measured. The plasma E_1, E_2, T, and LH concentrations were measured radioimmunologically using specific antisera, plasma F was determined by the competitive protein binding method, and the percentage of binding of E_1, E_2, T, and F was determined by equilibrium dialysis. The testes were normal in most of the patients with fatty liver and chronic hepatitis, but were small and soft in nine of the cirrhotic patients. Four fatty liver patients, 2 chronic hepatitis patients, and 12 cirrhotic patients complained of reduced potency or impotence, and gynecomastia was found in 10 of the cirrhotic patients. The plasma T concentrations were low normal in the fatty liver patients, reduced below normal levels in two hepatitis patients, and significantly reduced in the cirrhotic patients. The E_2 concentrations were normal in the fatty liver and chronic hepatitis patients and only slightly increased in the cirrhotic patients. The E_1 concentrations were also significantly increased in the cirrhotic patients and the plasma LH levels were increased by 50% over normal values in the hepatitis and cirrhosis patients. The F levels showed no dependence on age or hepatic disease. The percentage of bound T and E_2, but not that of E_1 or F, was also increased in the cirrhosis and hepatitis patients. In comparison with the young controls, the E_2/T ratio was moderately increased in the fatty liver patients, more markedly increased in the hepatitis patients, and greatly increased in the cirrhotic patients; these increases were even more marked

in the E_1/T ratio. It is suggested that the combination of elevated E_1 and E_2 and reduced T may be responsible for the gynecomastia and hypogonadism in chronic liver diseases. Furthermore, altered liver function may be responsible for alterations in steroid plasma concentrations and their binding to plasma proteins in both older men and liver disease patients.

6128 BILE ALCOHOL METABOLISM IN MAN: CONVERSION OF 5β-CHOLESTANE-3α,7α,12α,25-TETROL TO CHOLIC ACID. *(E.)* Salen, G.; Shefer, S.; Setoguchi, T.; Mosbach, E. H. (VA Hosp., East Orange, N. J.). *J. Clin. Invest.* 56(1):226-231, 1975.

To study the role of C_{25}-hydroxy bile alcohols as precursors of cholic acid, (G-^{3}H)5β-cholestane-3α,7α,12α,-25-tetrol was administered i.v. to two patients with cerebrotendinous xanthomatosis (CTX) and two normal individuals. One day after pulse labeling, radioactivity was present in the cholic acid isolated from the bile and feces of the CTX subjects and the bile of the normal controls. In the two controls, the specific activity decay curves of (G-^{3}H)cholic acid were exponential, and no traces of (G-^{3}H)5β-cholestane-3α,7α,12α,25-tetrol were detected. In contrast, appreciable quantities of labeled 5β-cholestane-3α,7α, 12α,25-tetrol were present in the bile and feces of the CTX patients. The specific activity *versus* time curves of fecal (G-^{3}H)5β-cholestane-3α,7α,12α,25-tetrol and (G-^{3}H)cholic acid showed a precursor-product relation in both groups of subjects. Although these results suggest that 5β-cholestane-3α,7α,12α,25-tetrol may be a precursor of cholic acid in man, the possibility that C_{26}-hydroxy intermediates represent the normal pathway cannot be excluded. The results also support the hypothesis that the genetic abnormality in CTX results in impaired bile acid synthesis, specifically, incomplete degradation of the cholesterol side chain.

6129 SELECTIVE LIGATION OF THE HEPATIC ARTERY FOR TRAUMA OF THE LIVER. *(E.)* Aaron, S.; Fulton, R. L.; Mays, E. T. (Univ. Louisville Sch. Med., Ky.). *Surg. Gynecol. Obstet.* 141(2):187-189, 1975.

The role of ligation of the hepatic artery in obtaining hepatic hemostasis is described in 60 patients over a 12-yr period. The right ramus was ligated in 36 patients, the left ramus in 15 patients, and the common hepatic artery in nine patients. In 55 patients, the artery was ligated on the day of injury at the time of initial laparotomy. Hemostasis and improved hemodynamic status were immediate and permanent in 49 patients. Six patients died in the operating room or immediately after laparotomy from head injury or uncontrolled hemorrhage from nonhepatic sites. Five patients required multiple laparotomies, and hemostasis was achieved in all cases after hepatic artery ligation. In three patients who had hepatic resection as the primary method of treating the hepatic wound, hemorrhage from the liver was profuse following resection. This postresection bleeding was controlled by hepatic artery ligation, and all patients survived. In a fourth patient, who had a severed left lateral segment, left hepatic artery ligation was used in conjunction with resection to successfully control hemorrhage. Seventeen patients died, including two with hemorrhage from perihepatic sites. The most common cause of death was sepsis, primarily subphrenic and pulmonary; six patients died from events unrelated to liver trauma. Hepatic insufficiency did not occur in survivors. It is concluded that ligation of the hepatic artery is an effective method of achieving hepatic hemostasis; it is simple, rapid, and universally applicable with predictable sequelae.

6130 REGULATION OF THE LIVER AND BLOOD PRESSURE. *(Ger.)* Wernze, H.; Brachtel, D.; Spech, H. J. (Med. Clin., Univ. Wurzburg, Germany). *Z. Gastroenterol.* 13(suppl.):120-132, 1975.

6131 HEPATOCELLULAR CARCINOMA IN JAPAN. *(Ger.)* Hattori, N.; Okazaki, N.; Ohno, T.; Mukojima, T.; Ohkura, H.; Araki, E.; Kitaoka, H., Hasegawa, H. (Nat. Cancer Cent. Hosp., Tokyo, Japan). *Leber Magen Darm* 5(2):58-61, 1975.

6132 MULTIPLE LIVER ABSCESSES WITH SPONTANEOUS PERFORATION INTO THE ABDOMINAL CAVITY NEAR THE BILE DUCT CLOSURE: OBSERVED BY PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY. *(Ger.)* Thammig, R.; Loncarevic, F.; Kloss, J. (St. Marien Hosp., Frankfurt/Main, Germany). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 122(1):81-82, 1975.

6133 INTERACTION BETWEEN THE BASIC POLYPEPTIDE TRASYLOL AND PROTEOGLYCANS. II: THE LIVER. *(Ger.)* Heine, H.; Eisenbach, J.; St. Gericke, D.; Forster, F. J.; Salzer, M. (Cent. Morphol., Univ. Frankfurt/Main, Germany). *Med. Welt* 26(16):753-758, 1975.

6134 METABOLISM, HEPATOLOGY AND ENDOCRINOLOGY. *(Ger.)* Schwandt, P. (Med. Clin. I, Univ. Munich, Germany). *Fortschr. Med.* 93(8):394-396, 1975.

6135 PHYSIOPATHOLOGICAL AND CLINICAL CONSIDERATIONS IN FOUR CASES OF INTRAHEPATIC CHOLESTASIS: UNCERTAIN ETIOLOGY AND SURGICAL INTERVENTION. Podda, M.; Tritapepe, R.; Ferla, G. F.; Bellomi, M.; Fichera, G. (Surg. Clin. I, Univ. Milan, Italy). *Arch. Ital. Chir.* 98(2):117-134, 1974.

6136 GIGANTIC CAVERNOUS HEMANGIOMA OF THE LIVER. *(Rus.)* Grinberg, S. B. (Tselinograd Med. Inst., USSR). *Vestn. Khir.* 114(4):117, 1975.

6137 CLINICAL SIGNIFICANCE OF DYSPROTEINEMIA IN CERTAIN DISEASES OF THE LIVER. *(Rus.)* Tsykin, D. B. (I. I. Mechnikov Sanit. Hyg. Med. Inst., Leningrad, USSR). *Klin. Med.* 53(1):67-71, 1975.

6138 PRIMITIVE CARCINOMA OF THE LIVER AND THE AUSTRALIA ANTIGEN: REPORT OF A CASE WITHOUT CIRRHOSIS. *(Sp.)* Garcia Bengoechea, M.; Lanas Larranaga, E.; Ortega Vega, J. (Our Lady of Aranzazu Resid. Sanit., San Sebastian, Spain). *Rev. Clin. Esp.* 134(5):495-500, 1974.

6139 PROTEIN SYNTHESIS AND EXCRETION BY THE LIVER IN COMPLICATIONS RESULTING FROM BLOOD TRANSFUSIONS. *(Rus.)* Rzhanovich, A. P.; Skachilova, N. N.; Vinogradova, I. L. (Cent. Inst. Hematol. Blood Transfus., Moscow, USSR). *Klin. Med. (Mosk.)* 53(3):105-109, 1975.

6140 MINERAL METABOLISM AND THE LIVER IN OBESITY. *(Rus.)* Kovaleva, N. B. (Sverdlovsk Med. Inst., USSR). *Klin. Med. (Mosk.)* 53(3):113-116, 1975.

6141 CURRENT THERAPY AND RECENT ADVANCES IN HEPATOLOGY. *(Fr.)* Darnis, F. (Beaujon Hosp., Clichy, France). *Cah. Med.* 15(9):559-565, 1974.

6142 HEPATOMAS: FREQUENCY AND MORPHOLOGICAL FEATURES IN 3700 AUTOPSIES. *(Fr.)* Brocheriou, C.; Auriol, M.; Ajebo, M.; Chomette, G. (Pitie-Salpetriere Hosp. Group, Paris, France). *Ann. Med. Interne (Paris)* 126(4):265-268, 1975.

6143 PRIMARY LIVER CANCER AND HEPATITIS B ANTIGEN (AUSTRALIA ANTIGEN): EPIDEMIOLOGICAL AND GENETIC STUDIES. *(Fr.)* Payet, M.; Saimot, G.; Brochard, C.; Coulaud, J. P.; Pasticier, A. (Claude-Bernard Hosp., Paris, France). *Ann. Med. Interne (Paris)* 126(4):269-271, 1975.

6144 HEPATITIS, INFECTIOUS MONONUCLEOSIS, AND EPSTEIN-BARR VIRUS. A CASE REPORT. *(Fl.)* Vadasz, T. A.; De Groote, J. (St-Joseph Hosp., Veghel, Belgium). *Tijdschr. Gastroenterol.* 17(2):115-118, 1974.

6145 α_1-ANTITRYPSIN. *(Fr.)* Nathanson, M. (no affil.). *Med. Infant.* 81(8):887-894, 1974.

6146 PRIMARY LIVER SARCOMAS. *(Rus.)* Kosarev, V. A.; Nikolaiev, A. V.; Ovchininskii, N. N. (no affil.). *Klin. Med.* 53(4):127-129, 1975.

6147 STUDIES OF POST-HEPARIN MONOGLYCERIDE LIPASE BY A NEW PHOTOMETRIC METHOD IN PATIENTS WITH LIVER DISEASE. *(Ger.)* Hansen, W. (2nd Med. Clin., Tech. Univ., Munich, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:494-496, 1974.

6148 COMPARISON OF THE PROTHROMBIN ACTIVITY AND CONCENTRATION IN VARIOUS CONDITIONS IN WHICH LIVER FUNCTION IS IMPAIRED. *(Ger.)* Oehler, G.; Bischoff, B.; Henk, R.; Bleyl, H. (Cent. Intern. Med., Giessen Univ., Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:1452-1456, 1974.

6149 THE CLINICAL AND IMMUNOLOGICAL DIAGNOSIS OF HEPATOCELLULAR CARCINOMA IN CHILDREN. *(Rus.)* Ionov, A. A.; Okulov, V. B.; Skachkov, A. P. (N.N. Petrov Res. Inst. Oncol., Leningrad, USSR). *Pediatriia* (3):81-83, 1975.

6150 THE ACTIVITY OF BENZPYRENE HYDROXYLASE IN HUMAN LIVER BIOPSIES IN VITRO AND ITS RELATION TO THE RATE OF GLYCODIAZINE EXCRETION IN VIVO. *(Ger.)* Held, H.; Schoene, B.; Laar, H. J.; Fleischmann, R. (Med. Clin., Univ. Tubingen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:501-503, 1974.

6151 RESECTION OF THE RIGHT LOBE OF THE LIVER IN PRIMARY LIVER CANCER. *(Rus.)* Zatolokin, V. D. (Kursk Med. Inst., USSR). *Klin. Khir.* (4):41-44, 1975.

6152 FROM EXPLORATION TO HEPATECTOMY IN LOCALIZED DISORDERS OF THE LIVER: 32 HEPATECTOMIES PERFORMED IN FOUR YEARS. *(Fr.)* Doutre, L. P.; Perissat, J.; Gouffrant, J. M.; Bobois, J. P.; Diard, F. (no affil.). *Bordeaux Med.* 7(16):2339-2346, 1974.

6153 PULMONARY PATHOLOGY IN SOME CHRONIC EXPERIMENTAL AND HUMAN LIVER DISEASES. *(Fr.)* Laky, D. (no affil.). *Poumon Coeur* 30(3):191-198, 1974.

6154 ASPIRIN AND LIVER FUNCTION. *(Fr.)* Lery, N.; Faivre, J.; Faivre, M.; Ducluzeau, R.; Paliard, P. (Edouard Herriot Hosp., Lyon, France). *Eur. J. Toxicol. Environ. Hyg.* 7(5-6):282-288, 1974.

6155 COMPARATIVE CHARACTERISTIC OF THE RADIOISOTOPIC AND BIOCHEMICAL METHODS OF INVESTIGATION OF LIVER FUNCTION IN HYPERTENSIVE DISEASE AND ATHEROSCLEROSIS. *(Rus.)* Fesenko, I. P. (Second Ther. Ward, Chernigov Reg. Hosp., USSR). *Vrach. Delo* 616(1):42-44, 1975.

6156 CHARACTERISTICS OF BILIRUBIN METABOLISM AND BILE MICROFLORA IN PATIENTS WITH CHRONIC LEUKOSES AND LYMPHOGRANULOMATOSIS. *(Rus.)* Sarnitskii, I. P.; Ishchenko, V. T.; Fomenko, T. L. (Dept. Hematol. Blood Transfusion, Kiev Inst. Postgrad. Training Phys., USSR). *Vrach. Delo* 616(1):73-77, 1975.

6157 THE NATURE OF AND CHANGES IN THE LESIONS OF HEPATIC PORPHYRIA. *(Sp.)* Perez, D. A. S. (1st. October Munic. Hosp., Madrid, Spain). *Clin. Eur.* 13(3):193-202, 1974.

6158 CURRENT TREATMENT OF HEPATIC AMEBIASIS. *(Fr.)* Laverdant, C.; Essioux, H.; Kassem, A. (Begin Military Hosp., Saint-Mande, France). *Arch. Fr. Mal. App. Dig.* 63(8):605-610, 1974.

6159 JAUNDICE ASSOCIATED WITH PREGNANCY. *(Fr.)* Frexinos, J.; Arany, Y. (Purpan Hosp., Toulouse, France). *Arch. Fr. Mal. App. Dig.* 63(8): 651-662, 1974.

6160 HEPATIC CARCINOSARCOMA: ONE CASE. *(Sp.)* Fundora Mayor, I.; Paramio Ruibal, A. (Manuel Fajardo Hosp., Luba, Spain). *Rev. Cub. Cir.* 13(6):691-697, 1974.

6161 HEPATIC ENCEPHALOPATHY AND MAGNESIUM DEFICIENCY STATES. *(Pol.)* Pawlak, J. (J. Brudzinski Hosp., Lodz, Poland). *Wiad. Lek.* 28(2): 103-106, 1975.

6162 A CASE OF RECURRENT TUMOR IN THE GREATER OMENTUM SUCCESSFULLY REMOVED ABOUT 9 YEARS AFTER RESECTION OF HEPATOMA IN THE LIVER. *(Jpn.)* Konno, T.; Arita, T.; Kimura, A.; Abe, K. (Ooita-Ishikairitsu Hosp., Ooita, Japan). *Gan No Rinsho* 20(11):992-998, 1974.

6163 TREATMENT OF FATTY LIVER. *(Ger.)* Phlippen, R. (Med. Clin., Univ. Cologne, Germany). *Med. Welt* 25(46):1916-1920, 1974.

6164 TREATMENT OF HEMOCHROMATOSIS AND PORPHYRIA. *(Ger.)* Strohmeyer, G. (2nd Med. Clin., Univ. Dusseldorf, Germany). *Med. Welt* 25(46):1911-1915, 1974.

6165 DIET IN LIVER DISEASES. *(Ger.)* Frotz, H. (Med. Clin., Univ. Cologne, Germany). *Med. Welt* 25(46):1927-1930, 1974.

6166 DIFFERENTIAL DIAGNOSIS OF RIGHT-SIDED DIAPHRAGMATIC HERNIA WITH PROLAPSE OF THE LIVER USING ANGIOGRAPHY. *(Ger.)* Bucheler, E. (Radiol. Clin., Univ. Bonn, Germany). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 121(3):296-302, 1974.

6167 TREATMENT OF ACUTE HEPATIC NECROSIS BY LIVER TRANSPLANTATION: COMPARATIVE STUDY OF ORTHOTOPIC AND HETEROTOPIC TRANSPLANTS. *(Fr.)* Huguet, C.; Bloch, P.; Opolon, P.; Conard, J.; Lavallard, M. C.; Morisot, P.; Cornier, E.; Nordlinger, B.; Hamani, A. (St. Antoine Hosp., Paris, France). *J. Chir. (Paris)* 108(4):397-406, 1974.

6168 INTRAHEPATIC DISEASES OF THE BILIARY TRACT BASED ON BIOPSY FINDINGS. *(Ger.)* Roschlau, G. (Carl Gustav Carus Med. Acad., Dresden, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):125-128, 1974.

6169 CIRCULATORY DISORDERS OF THE LIVER: FORMS AND CONSEQUENCES FROM A MORPHOLOGICAL VIEWPOINT. *(Ger.)* Altmann, H. W. (Pathol. Inst., Univ. Wurzburg, Germany). *Z. Gastroenterol.* 13(suppl):77-103, 1975.

6170 PRIMARY HEPATIC ABSCESSES: STUDY OF NINE CASES. *(Sp.)* Novo Prego, C.; Merino Angulo, J.; Diego Marin, J.; Martinez Lopez de Letona, J. (Puerta de Hierro Clin., Madrid, Spain). *Rev. Clin. Esp.* 135(1):59-64, 1974.

6171 HEPATIC METASTASES IN GASTROINTESTINAL CANCER: FREQUENCY, PROGNOSIS AND POSSIBILITIES FOR SURGERY. *(Fr.)* Huguier, M. (Rothchild Hosp., Paris, France). *Concours Med.* 97(12):1977-1984, 1975.

6172 HEMANGIOMA OF THE LIVER WITH CONSIDERABLE ARTERIOVENOUS SHUNT IN A TWO MONTH OLD BOY. *(Ger.)* Globl, H. J.; Kaufmann, H. J. (Child. Hosp., Philadelphia, Pa.). *Z. Kinderchir. Grenzgeb.* 15(2): 242-244, 1974.

6173 WHAT IS GUARANTEED IN SHORT-TERM TREATMENT OF PATIENTS WITH LIVER OR GALLBLADDER DISEASE? *(Ger.)* Bode, J. C.; Martini, G. A. (Med. Clin., Univ. Marburg/Lahn, Germany). *Internist* 15(12):605-614, 1974.

6174 ACUTE HEPATIC PORPHYRIA. *(Nor.)* Stenerud, O. (Dept. VII, Internal Med., Ulleval Hosp., Norway). *Tidsskr. Nor. Laegeforen.* 95(10):590-592, 1975.

6175 CRITICAL EVALUATION OF THE VALUE OF HEPATOPROTECTIVE THERAPY. *(It.)* Rinetti, M.; Dei Cas, L. (Inst. Gen. Clin. Med. Med. Ther. I, Parma, Italy). *Minerva Gastroenterol.* 20(3):90-96, 1974.

6176 THE PATHOGENESIS OF PELIOSIS HEPATITIS. *(Por.)* da Silva Horta, J.; da Silva Horta, M. E. (Fac. Med., Lisbon, Portugal). *J. Soc. Cien., Med. Lisboa* 139(1):9-28, 1975.

6177 BILIRUBIN METABOLISM. II. UPTAKE OF BILIRUBIN BY THE LIVER AND THE CONJUGATION OF BILIRUBIN. *(Sp.)* Candel Monserrate, I. (Fac. Med., Univ. Complutense, Madrid, Spain). *Rev. Esp. Enferm. Appar. Dig.* 45(4):433-448, 1975.

6178 THE ACTION OF Chelidonium, Curcuma, ABSINTHE, AND Carduus marianus ON BILIARY AND PANCREATIC SECRETION IN HEPATIC PATHOLOGY. *(Ger.)* Baumann, J. C. (Auguste-Victoria Munic. Hosp., Berlin-Schoneberg, Germany). *Med. Monatschr.* 29(4):173-180, 1975.

6179 VARIATIONS IN LIVER METABOLISM DURING STREPTOKINASE-INDUCED THROMBOLYSIS THERAPY. *(Ger.)* Bruhn, H. D. (no affil.). *Thromb. Diath. Haemorrh.* (Suppl. 61):167-170, 1975.

6180 CYTOHISTOLOGICAL ASPECTS OF THE LIVER IN WILSON'S DISEASE. *(It.)* Cecio, A.; Del Vecchio Blanco, C.; Di Simone, A.; Caporaso, N.; de Girolamo, G. (Fac. Med. I, Univ. Naples, Italy). *Boll. Soc. Ital. Biol. Sper.* 50(18):1512-1515, 1974.

6181 LIPOPROTEIN-X (LP-X) IN THE DIAGNOSIS OF INTRAPATHIC CHOLESTASIS (PREVENTIVE NOTE). *(It.)* Colalongo, G.; Nubile, G.; Fiorentini, G. P.; Tabacchi, L.; Beltrandi, E. (Sch. Gen. Pathol., Univ. Chieti, Italy). *Boll. Soc. Ital. Biol. Sper.* 50(18): 1440-1445, 1974.

6182 EXPLORATION OF HEPATOBILIARY FUNCTION WITH ^{131}I-LABELED BSP. I) CLEARANCE OF [^{131}I]BSP. *(It.)* Quattrocchi, G.; Squillaci, S.; Smedile, G.; Grasso, E. (Inst. Spec. Med. Pathol. Clin. Methodol., Univ. Messina, Italy). *Boll. Soc. Ital. Biol. Sper.* 50(13):919-923, 1974.

6183 EXPLORATION OF HEPATOBILIARY FUNCTION WITH ^{131}I-LABELED BSP. II) DYNAMIC HEPATOBILIARY SCINTIGRAPHY. *(It.)* Quattrocchi, G.; Squillaci, S.; Grasso, E.; Arcoraci, A. (Inst. Spec. Med. Pathol., Univ. Messina, Italy). *Boll. Soc. Ital. Biol. Sper.* 50(13):924-929, 1974.

6184 HEPATIC LESIONS IN PRIMARY MYELOSCLEROSIS IN AN ADULT. *(Fr.)* Waitz, R.; Mayer, S.; Mayer, G.; Oberling, F. (no affil.). *Bull. Acad. Natl. Med. Paris* 159(1):71-77, 1975.

6185 SURGICAL THERAPY AND INTRAOPERATIVE DIAGNOSIS OF BILIARY TRACT DISEASE. *(Ger.)* Spohn, K.; Fux, H. D.; Muller-Kluge, M.; Wallenstein, F.; Tewes, G. (Munic. Hosp., Karlsruhe, Germany). *Therapiewoche* 25(9):1033-1047, 1975.

6186 EXPERIENCE IN LONG-TERM THERAPY OF LIVER DISEASES WITH A COMBINED PREPARATION OF HEPASTERIL^{R}-INJECTION AMPOULE. *(Ger.)* Ohlenschlager, G. (no affil.). *Therapiewoche* 25(6):632-639, 1975.

6187 LONG-TERM TREATMENT OF CHRONIC HEPATITIS AND LIVER CIRRHOSIS. *(Ger.)* Pusch, H. J.; Ullmann, W. (Med. Polyclin., Univ. Wurzburg, Germany). *Med. Klin.* 70(15):674-681, 1975.

6188 STUDY ON FIBRINOLYTIC PHENOMENON IN THE BLOOD OF HEPATOBILIARY DISEASES. *(Jpn.)* Yamaguchi, M. (Sch. Med., Yoho Univ., Japan). *Arch. Jpn. Chir.* 43(5):359-372, 1974.

6189 ALPHA$_1$-FETOPROTEIN IN THE CHILD. *(Fr.)* Buffe, M. D.; Rimbaut, M. C. (Inst. Res. Cancer, Villejuif, France). *Bordeaux Med.* 8(6):671-675, 1975.

6190 INTRAHEPATIC BILIARY ATRESIA IN DOWN'S SYNDROME. *(E.)* Puri, P.; Guiney, E. J. (no affiliation given). *J. Pediatr. Surg.* 10(3):423-424, 1975.

6191 SOLITARY LIVER NODULES. *(E.)* Fisher, A. W. F.; Curry, B.; Jacques, J. (Faculty Medicine, Univ. Calgary, Calgary, Alta., Canada). *Can. Med. Assoc. J.* 112(10):1196-1200, 1975.

6192 NON-CIRRHOTIC PORTAL HYPERTENSION IN FELTY'S SYNDROME. *(E.)* Sweeney, E. C.; (R. Postgrad. Med. Sch., London, England). *Irish J. Med. Sci.* 144(4):172-174, 1975.

6193 LIVER TRAUMA IN CHILDREN. *(E.)* Suson, E. M.; Klotz, Jr., D.; Kottmeier, P. K. (New York Downstate Med. Cent., Brooklyn). *J. Pediatr. Surg.* 10(3):411-417, 1975.

6194 HEPATIC CHOLESTEROL SYNTHESIS IN MAN: EFFECT OF DIAZEPAM AND OTHER DRUGS. *(E.)* Orlandi, F.; Bamonti, F.; Dini, M.; Koch, M.; Jezequel, A. M. (Sch. Med., Univ. Ancona, Italy). *Eur. J. Clin. Invest.* 5(2):139-146, 1975.

6195 HEPATOCELLULAR CARCINOMA: RELATION TO ALCOHOL, HB-ANTIGEN AND ALPHA-FETOPROTEIN. *(E.)* Dourdourekas, D.; Villa, F.; Szanto, P. B.; Steigmann, F. (Hektoen Inst. Med. Res., Chicago, Ill.). *Am. J. Gastroenterol.* 63(4):307-311, 1975.

6196 THERAPEUTIC APPROACHES TO HEPATOMA. *(E.)* Falkson, G. (H. F. Verwoerd Hosp., Pretoria, South Africa). *Cancer Treat. Rev.* 2(1):73-76, 1975.

6197 EPIDEMIOLOGIC INVESTIGATION OF REYE SYNDROME OUTBREAK IN ST. LOUIS SCHOOLS. *(E.)* Stechenberg, B. W.; Keating, J. P.; Chang, M.; Haymond, M. W.; DeVivo, D.; Feigin, R. D. (St. Louis, Mo.). *J. Pediatr.* 86(6):974, 1975.

6198 SPONTANEOUS RUPTURE OF THE LIVER AND THE SPLEEN: REPORT OF A SUCCESSFULLY TREATED CASE WITH IMMUNOPATHOLOGIC REACTION. *(E.)* Huttunen, R.; Seppala, A.; Mokka, R.; Larmi, T. K. I. (Dep. Surg., Univ. Oulu, Finland). *Surgery* 77(5):722-725,1975.

6199 PRIMARY ENDODERMAL SINUS (YOLK SAC) TUMOR OF THE LIVER. FIRST REPORTED CASE. *(E.)* Hart, W. R. (Dep. Pathol., Univ. South. Calif., Los Angeles). *Cancer* 35(5):1453-1458, 1975.

6200 FOCAL NODULAR HYPERPLASIA OF THE LIVER: A FURTHER CONTRIBUTION. *(E.)* O'Reilly, K. (Mater Misericordia Hosp., Brisbane, England). *Aust. N.Z. J. Surg.* 45(1):76-77, 1975.

6201 CHOLANGIOCARCINOMA IN A PATIENT PREVIOUSLY GIVEN THOROTRAST. *(E.)* Johnson, P. K.; Babb, R. R. (Palo Alto Med. Clin., Calif.). *Am. J. Dig. Dis.* 20(4):384-390, 1975.

6202 LIVER LIPIDS IN PATIENTS WITH ENDOGENOUS HYPERTRIGLYCERIDEMIA. *(E.)* Maruhama, Y.; Yanbe, A.; Tadaki, H.; Ohtsuki, M.; Ohneda, A.; Abe, R.; Yamagata, S. (Tohoku Univ. Sch. Med., Sendai, Japan). *Tohoku J. Exp. Med.* 114(3):247-252, 1974.

6203 HEPATOSTOMY FOR CENTRAL HEPATIC HEMATOMAS. *(E.)* Gewertz, B. L.; Olsen, W. R. (Univ. Michigan Med. Cent., Ann Arbor). *J. Trauma* 15(4): 271-274, 1975.

6204 BARIUM ENEMA FINDINGS IN TYPE I HEPATORENAL GLYCOGEN STORAGE DISEASE. *(E.)* Fellows, R. A.; Berdon, W. E.; Baker, D. H.; Harris, R. (Columbia-Presbyterian Med. Cent., New York, N.Y.). *Pediatr. Radiol.* 3(2):75-77, 1975.

6205 PHYSIOLOGIC CONSEQUENCES OF HEPATIC LOBECTOMY IN MAN. *(E.)* Mays, E. T. (Univ. Louisville Sch. Med., Ky.). *South. Med. J.* 68(4): 399-406, 1975.

6206 PERCUTANEOUS LONG-TERM HEPATIC ARTERY INFUSION FOR PRIMARY AND METASTATIC CANCER IN THE LIVER. METHOD IS USEFUL WHERE INITIAL OPERATIVE PLACEMENT IS NOT POSSIBLE OR WHERE PATIENT REFUSES SURGERY. *(E.)* Mansour, E. G. (Dep. Surg., Case Western Reserve Univ., Cleveland, Ohio). *R.I. Med. J.* 58(3):91-93, 1975.

6207 DOUBLE-BLIND STUDY OF A TOTAL LIVER EXTRACT IN PATIENTS WITH HEPATIC DYSFUNCTION. *(E.)* Preziosi, P.; Nistico, G.; Marano, V. (Inst. Pharmacol., Univ. Naples, Italy). *Int. J. Clin. Pharmacol. Biopharm.* 3(11):210-215, 1975.

6208 PAEDIATRIC LIVER DISEASE. *(E.)* Mowat, A. P. (King's Coll. Hosp. Med. Sch., London, England). *Br. J. Hosp. Med.* 13(1):55-56, 61-64, 1975.

6209 A LARGE HEPATOMA WITHOUT APPARENT ABNORMALITY ON LAPAROTOMY. *(E.)* Tsuzuki, T.; Hasumi, A.; Suguira, Y.; Tsuchiya, M.; Kamegaya, K.; Hiramatsu, K. (Sch. Med., Keio Univ., Tokyo, Japan). *Keio J. Med.* 22(3):137-142, 1973.

See also, 5420, 5616, 5620, 5627, 5630, 5632, 5634, 5636, 5637, 5642, 5666, 5668, 5669, 5672, 5677, 5682, 5696, 5699, 5700, 5816, 6041, 6046, 6211, 6220, 6248, 6304, 6329, 6330, 6344, 6356, 6367, 6378, 6381, 6387, 6388, 6405, 6419, 6427, 6428, 6497, 6506, 6519, 6543, 6549, 6551, 6552, 6556, 6558, 6570, 6571.

LIVER AND BILIARY TRACT

Hyperbilirubinemic States

6210 SLOW INFUSION CHOLANGIOGRAPHY IN PATIENTS WITH JAUNDICE. *(Ger.)* Fuchs, W. A.; Preisig, R. (Inselspital, Bern, Switzerland). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 122(2):148-151, 1975.

A solution of 40 ml (20 g) ioglycamide in 500 ml physiological saline was infused dropwise over a 10 hr period, i.e. at a rate of about 35 mg/min, in 30 fasting patients (19 men and 11 women, aged 20-77 yr) with jaundice caused by a variety of hepatobiliary diseases. All patients had serum bilirubin levels of 4 mg/100 ml or more (1.4-13.6 mg/100 ml), and all were suspected of having extrahepatic biliary obstructions. X-rays were taken with the patients in a prone position, using 75 kilovolts or less, before and 15 min after p.o. administration of 1 g lecithin suspended in 25 ml of an emulsion of egg yolk and glycerol. Tomography was routinely used for better visualization of the gallbladder and bile ducts which, in many cases, filled only slightly. The diagnosis was confirmed by surgical exploration in 18 cases, by histological findings in association with the clinical course in 11 cases, and at autopsy in one case. Slow infusion cholangiography made it possible to establish a correct radiological diagnosis in 11 of 30 patients (38%). While visualization of the extrahepatic bile ducts was good enough to establish a diagnosis in only 2 of 15 patients with extrahepatic biliary obstructions (13%), a radiological diagnosis was made in 9 of 15 patients with parenchymal liver disease (60%).

6211 LONG-TERM PROGNOSIS IN INTRAHEPATIC BILIARY ATRESIA AND IN THE SO-CALLED NEONATAL HEPATITIS. *(Ger.)* Osswald, P.; Gathmann, H.; Muller, G. (Child. Clin., Univ. Tubingen, Germany). *Monatschr. Kinderheilkd.* 123(5):445-447, 1975.

Follow-ups were performed on 20 infants with histologically confirmed neonatal hepatitis and on nine with hypoplasia of the intrahepatic bile ducts.

While rapid regression of the symptoms of neonatal hepatitis was a favorable sign, laboratory examinations performed in initial stages of the disease were of little value in establishing a prognosis. Of the 20 infants (16 boys and four girls) with neonatal hepatitis, nine showed histological evidence of giant cell transformation. In contrast to other reports in the literature, the symptoms and course of infants with giant cell transformation did not differ appreciably from those in the remaining 11 patients. Jaundice developed by age 2 weeks in 2/3 of these patients and, with one exception, maximum total bilirubin values ranged from 4.8 to 18.4 mg/100 ml. In six cases, neonatal hepatitis was associated with other diseases (toxoplasmosis, rubella, hepatitis B, α_1-antitrypsin deficiency and clinically diagnosed Edward's syndrome). Liver damage accompanied by clotting disorders, ascites, edema and, in some cases, in combination with intercurrent infection was the cause of death in the nine patients who died before their first birthday. Two other children died of hepatic coma and esophageal varices, resp., before 4 yr of age. Of these 20 children, only five showed no clinical evidence of residual liver damage at an average age of 6 yr, although the initial stages of cirrhosis were diagnosed histologically during the first four months of life, at which time bilirubin levels and all other liver function tests had normalized. Hypoplasia of the intrahepatic bile ducts, diagnosed in nine infants by histological findings on wedge liver biopsies, followed a benign course in four children; bilirubin levels and other liver function tests normalized by age 3-5 months and no residual clinical symptoms were apparent at ages 1.5 to 8 yr. In the remaining five cases, chronic cholestasis, increased portal fibrosis, and proliferation of the intralobular bile ducts developed along with a secondary disorder in lipid metabolism. The latter was accompanied by great increases in free cholesterol and phosphatide concentrations, a positive lipoprotein X test and significant increases in serum bile acid levels. Only 1/5 children developed

6212 USE OF OXYTOCIN AND INCIDENCE OF NEONATAL JAUNDICE. *(E.)* Chalmers, I.; Campbell, H.; Turnbull, A. C. (London Sch. Hyg. Trop. Med., England). *Brit. Med. J.* 2(5963)116-118, 1975.

A retrospective controlled study using data from the Cardiff Births Survey was performed to examine a possible relation between oxytocin administration to induce or accelerate labor and the subsequent development of neonatal jaundice. The study group consisted of 10,591 infants, or 61% of the total live births between 1970-1972, and 95% of those discharged from 3-8 days after delivery. Of the 10,591 infants, 970 (9.2%) had a measured plasma bilirubin of at least 17 μM/liter. Of the 3326 born after oxytocin administration, 412 (12.4%) became jaundiced. Of the 5896 born after spontaneous labor, 476 (8.1%) were affected. Of the 1369 born after amniotomy, 82 (6.0%) developed jaundice. Of the 7265 infants in the latter two groups (no oxytocin), 558 (7.7%) became significantly jaundiced, so that the relative risk of jaundice in an infant born after oxytocin administration compared to the risk in other infants was 1.6. However, analysis by gestational age at delivery, birth weight, Apgar score, length of labor, sedative and analgesic therapy during labor, and suppression of lactation showed that the association between jaundice and oxytocin held within all these categories except among small immature infants who are at high risk of jaundice in any case. The apparent protection against jaundice conferred by amniotomy *versus* spontaneous labor reflected the low incidence of amniotomy at the early gestational age (36 and 37 weeks) which showed a high overall incidence of jaundice. Therefore, the infants born after spontaneous labor and those born after amniotomy were comparable with respect to the incidence of jaundice. These results support the hypothesis that the increasing resort to induction and acceleration of labor has been associated with a higher incidence of neonatal jaundice. It is suggested that this may not be the only enzyme system which is affected.

6213 MICROHETEROGENEITY OF COMPLEX GLYCOSIDES OF BILIRUBIN FROM HUMAN BILE. *(E.)* Kuenzle, C. C. (Sch. Vet. Med., Univ. Zurich. Switzerland). *Experientia* 31(6):626-627, 1975.

A chromatographic system was developed to allow the preoperative separation of the aldobiouronide, hexuronosylhexuronide, and pseudoaldobiouronide conjugates of bilirubin on polyamide sheets. Bilirubin conjugates were isolated from human T-tube bile as their phenylazo derivatives, and the aldobiouronide, hexuronosylhexuronide, and pseudoaldobiouronide conjugates were obtained by column chromatography. These preparations were freed from contaminating celite, extracted with 1-butanol and dried. The residue was dissolved in methanol, streaked on polyamide sheets, and developed using a solvent system prepared from 4-methyl-2-penanone. Using this fractionation system, virtually identical chromatographic patterns were obtained for all three azo pigments. Three azo pigment subfractions were visualized in all three instances. Gross estimation of the subfractions gave an approximate intragroup ratio of 5:60:35. The system was not entirely satisfactory, however, in that the azo pigments of lowest mobility were incompletely separated from those of intermediate mobility; also, elution of the subfractions from the plates resulted in partial solubilization of the chromatographic support, thus contaminating the preparations with depolymerizing material. It is concluded that the aldobiouronic, hexuronosylhexuronic, and pseudoaldobiouronic acids involved in bilirubin conjugation are heterogeneous.

6214 FLUOROMETRIC DETERMINATION OF 'ALBUMIN-TITRATABLE BILIRUBIN' IN THE JAUNDICED NEONATE. *(Eng.)* McCluskey, S. B.; Storey, G. N. B.; Brown, G. K.; More, D. G.; O'Sullivan, W. J. (Royal Prince Alfred Hosp., Camperdown, N.S.W. 2050, Australia). *Clin. Chem.* 21(11):1638-1643, 1975.

A fluorometric technique is presented for the determination of albumin-titratable bilirubin in the jaundiced neonate. Although bilirubin alone has very little fluorescence, considerable emission is observed in the presence of albumin under acid conditions. Blood samples were obtained from umbilical cord veins or by heelprick from newborn infants. Blood from normal healthy women volunteers was collected by venipuncture. Albumin in the plasma samples was determined by a modified bromcresol green method or, in the case of albumin solutions, by its absorption at 279 nm. Total serum protein was measured by refractive index. Four glass tubes were set up for each blood sample, two containing 40 µl of saline solution and two containing 50 µl of normal human serum or a serum albumin solution (40 g/l). Plasma (10 µl) was pipetted into each tube. A standard bilirubin curve was set up in the range of 0 to 200 mg/l. A tube containing 40 µl of human serum and 10 µl of saline provide a correction factor for the endogenous bilirubin and albumin present in the normal human serum. Phosphoric acid (0.6 ml) was added to the standards and samples and mixed. At least 1 min after mixing, 3 ml of ascorbic acid reagent was added to each tube and mixed again. Fluorescence short-scan measurements were made within 2 hr in quartz cuvets at: excitation 435 nm, excitation slit 6 mm, emission 480 to 520 nm, and emission slit 12 mm. Analysis of the plasma sample alone and in the presence of excess human serum albumin solution appeared to reflect the bilirubin tightly bound to albumin and the total serum bilirubin, resp. The differences between these two values is the albumin-titratable bilirubin. A typical saturation effect was observed with considerable albumin-titratable bilirubin concentrations. In samples where the circulating bilirubin was strongly bound to endogenous albumin, no change was observed when exogenous albumin was added. This method appears to provide a useful supplement to currently available procedures for the assessment of the jaundiced neonate. It is easy to perform, requires only small amounts of plasma, and the results correlate well with the clinical picture.

6215 CALORIC INTAKE AND UNCONJUGATED HYPERBILIRUBINEMIA. *(E.)* Felsher, B. F.; Carpio, N. M. (VA Hosp., Long Beach, Calif.). *Gastroenterology* 69(1):42-47, 1975.

The caloric effect observed in Gilbert's syndrome was investigated to determine its relation to a higher prefasting serum bilirubin level or the underlying uridine diphosphate glycuronyltransferase (UDPG-T) deficiency. UDPG-T assays were performed on liver biopsies from patients with Gilbert's syndrome. Diet studies were performed on 10 patients with Gilbert's syndrome, in 7 patients with hemolysis, and in 13 normal subjects. The subjects fasted for 48 hr prior to measurement of the serum total and direct reacting bilirubin concentration. Two normal subjects were also treated with novobiocin prior to and while fasting. In patients with Gilbert's syndrome and in normal subjects the increase in total serum bilirubin after a 48-hr fast was statistically significant, but the absolute increase in serum bilirubin concentration after a fast was much greater in Gilbert's syndrome compared with normal subjects. In patients with hemolysis, the increase in mean serum bilirubin level after fasting, in spite of a higher mean prefasting level, was significantly less than that obtained in the group with Gilbert's syndrome and only slightly greater than in the normals. Disparate results were obtained in two additional patients with hemolysis and unconjugated hyperbilirubinemia. The reciprocal relations of caloric intake to the serum bilirubin level demonstrated by these patients more closely resembled that observed in the patients with Gilbert's syndrome than the other patients with hemolysis. A comparison of hepatic UDPG-T activities in all subjects showed that the enzymic activities in the two patients with hemolysis and an atypical caloric effect were reduced to levels comparable to Gilbert's syndrome. In two normal subjects, the serum total bilirubin concentrations increased, resp., 0.7 and 0.6 mg/100 ml after novobiocin, and 4.4 and 3.3 mg/100 ml after a combination of fasting and novobiocin. The serum conjugated fractions were not increased. The results indicate that unconjugated hyperbilirubinemia *per se* is not responsible for the exaggerated response in Gilbert's syndrome and suggest that the underlying reduction in UDPG-T activity is the responsible factor. Fasting appears to differentiate Gilbert's syndrome from hemolytic jaundice as well as from normal subjects, irrespective of the initial serum bilirubin concentration.

6216 GLUCOCORTICOIDS AND BILIRUBIN METABOLISM IN JAUNDICE. *(Pol.)* Pirwitz, B. (Clin. Gastroenterol. Metabol., Postgrad. Training Ctr. Phys., Warsaw, Poland). *Przegl. Lek.* 31(10):865-868, 1974.

See also, 5439, 5621, 5624.

LIVER AND BILIARY TRACT

Nonviral Hepatitis and Hepatic Injury

6217 ISONIAZID-ASSOCIATED HEPATITIS: SERUM ENZYME DETERMINATIONS AND HISTOLOGIC FEATURES. *(E.)* Lewis, J. E.; Mello, P.; Knauer, C. M. (Santa Clara Valley Med. Cent., San Jose, Calif.). *West. J. Med.* 122(5):371-376, 1975.

The frequency and severity of the physiologic, histologic, and symptomatic features of isoniazid-associated hepatitis were studied prospectively in 208 patients treated for up to 12 months with isoniazid for tuberculosis prophylaxis. General clin-

ical evaluations and SGOT evaluations were performed on all patients prior to the initiation of isoniazid treatment and 1, 2, 3, 6, 9, and 12 months thereafter. Serum bilirubin, alkaline phosphatase, SGPT, and hepatitis-associated antigen (HAA) were also measured in those with elevated SGOT levels. Elevated SGOT levels developed in 43% of the 47 adults who developed symptoms of hepatitis, 16% of the 109 asymptomatic patients, and 18% of the 142 patients without history of alcohol abuse. Six patients had elevated SGOT levels before initiation of isoniazid therapy, 12 patients with normal pretreatment SGOT levels had single or nonconsecutive elevations in SGOT during therapy, and 19 patients with normal pretreatment SGOT levels had two or more consecutive elevations in SGOT during therapy. The SGPT levels were elevated in eight of these patients, but alkaline phosphatase and bilirubin were generally within normal limits and HAA was negative. Percutaneous liver biopsy was done in eight patients with persistent SGOT elevation during isoniazid therapy; nonspecific hepatitis was evident in five, slight portal fibrosis and early bile duct proliferation were found in another, and mild to moderate microvesicular fatty metamorphosis were observed in the other two. Among the 33 isoniazid-treated children, SGOT abnormalities developed in two. It is recommended that monthly clinical evaluations be made in patients receiving isoniazid. In symptomatic patients, liver function tests should be performed and in cases of prolonged elevations in hepatic enzymes, bilirubin, and/or alkaline phosphatase, isoniazid administration should be discontinued. Liver biopsies may be indicated in these patients, particularly if the liver function parameters do not return to normal values after discontinuation of isoniazid.

6218 INCREASED INCIDENCE OF ISONIAZID HEPATITIS IN RAPID ACETYLATORS: POSSIBLE RELATION TO HYDRAZINE METABOLITES. *(E.)* Mitchell, J. R.; Thorgeirsson, U. P.; Black, M.; Timbrell, J. A.; Snodgrass, W. R.; Potter, W. Z.; Jollow, D. J.; Keiser, H. R. (Lab. Clin. Pharmacol., Natl. Heart Lung Inst., Bethesda, Md.). *Clin. Pharmacol. Ther.* 18(1):70-79, 1975.

To determine whether susceptibility to hepatic injury is correlated with the rapid acetylation of isoniazid, 26 non-Oriental patients who had recovered from probable or possible isoniazid hepatitis were genetically phenotyped as rapid or slow metabolizers of isoniazid, based on their ability to acylate orally administered sulfamethazine (40 mg/kg). Then, to determine whether other differences occur in the metabolism of isoniazid in rapid and low acetylators, radiolabeled isoniazid and acetylisoniazid (5 mg/kg) were given p.o. to seven adult male volunteers, and the urinary metabolites were isolated and identified by paper chromatography and mass spectrometry. Among the 21 subjects who had previously suffered probable isoniazid liver injury, 86% displayed the rapid phenotype for sulfamethazine acetylation. Of the five subjects who had suffered possible isoniazid liver injury, 60% were rapid acetylators; the expected frequency was 45%. Analysis of the urinary isoniazid metabolites in the second study showed that the amounts of isoniazid hydrozones excreted approximately paralleled the isoniazid excretion in both slow and rapid acetylators. However, the excretion of acetylisoniazid and isonicotinic acid and its glycine conjugate were inversely related to the amount of isoniazid excreted. Acetylisoniazid metabolism was the same in both slow and rapid acetylators, no hydrazones being formed after acetylisoniazid administration. It is suggested that the release of the hepatotoxic hydrazino moiety of isoniazid in man is responsible for isoniazid liver injury.

6219 QUINIDINE HEPATITIS. *(E.)* Handler, S. D.; Hirsch, N. R.; Haas, K.; Davidson, F. Z. (Montefiore Hosp. Med. Cent., Bronx, N.Y.). *Arch. Intern. Med.* 135(6):871-872, 1975.

A 72 yr-old woman receiving quinidine (200 mg/6 hr) for 16 months developed abnormal levels of SGOT, alkaline phosphatase and LDH, which returned to normal upon discontinuation of the drug. Upon challenge with 200 mg of quinidine, the levels became abnormal. Prior to therapy, SGOT was within normal limits (10-50 mIU/ml), but 7 months after initiation, SGOT was 367 mIU/ml. During the following 9 months, SGOT ranged from 490 to 795 mIU/ml and the patient suffered anorexia and a 4.5 kg weight loss. At this time, other medications included diphenhydramine, triamterene, hydrochlorothiazide, meprobamate, estrogen, digoxin, nitroglycerin and Ornade. All medications, except digoxin, were discontinued 2 weeks prior to admission. At that time, SGOT was greater than 300 mIU/ml; alkaline phosphatase was raised to 110 mIU/ml, and LDH was raised to 417 mIU/ml. On admission, SGOT was 155 mIU/ml; alkaline phosphatase was 93 mIU/ml and LDH was 220 mIU/ml. Counter immunoelectrophoresis for hepatitis B antigen and antibody was negative. Percutaneous liver biopsy revealed focal areas of hepatocellular necrosis and replacement by Kupffer cells, suggesting active, acute nonspecific hepatitis. After liver function tests returned to normal for 2 weeks, 200 mg of quinidine was given as a challenge. Within 24 hrs, anorexia, vomiting and weakness developed and SGOT climbed to 110 mIU/ml by 72 hrs. Quinidine appears to have a hepatotoxic effect which currently must be labeled as idiopathic.

6220 IS LIVER INJURY PRODUCED BY HIGH-CALORIE INFUSION THERAPY? *(Ger.)* Pfeffer, H. (Surg. Clin., Univ. Giessen, Germany). *Diagnostik* 8(8):307-311, 1975.

After a 12-14 hr fast, five groups of metabolically normal male and female mongrel dogs were infused for 6 hr/day over a 4-day period with the following: (1) 1 ml/kg/hr of 0.9% NaCl; (2) 2 ml/kg/hr of a high-calorie (2.216 kcal/kg/hr) solution containing glucose (0.06 g/kg/hr) + fructose (0.14 g/kg/hr) + xylotol (0.20 g/kg/hr) + ethanol (0.08 g/kg/hr); (3) 4 ml/kg/hr of a high-calorie (4.432 kcal/kg/hr) solution containing glucose (0.20 g/kg/hr) + fructose (0.28 g/kg/hr) + xylitol (0.40 g/kg/hr) + ethanol (0.16 g/kg/hr); (4) 4 ml/kg/hr of the high-calorie solution + 2.5 mg ATP (1.185 mg PO_4^{-3}/kg/hr); and

(5) 4 ml/kg/hr of the high-calorie solution + 0.72 ml Sörensen buffer (14.400 mg/kg/hr PO_4^{-3}). High-calorie solutions also contained Na^+, K^+, Mg^{++}, and Cl^-. On days 1 and 3, the activities of the following enzymes were determined on blood samples taken before infusion and 1, 2, 4, and 8 hr thereafter: SGOT, SGPT, alkaline phosphatase, LDH, leucine aminopeptidase (LAP), γ-glutamyl transpeptidase and cholinesterase. Blood samples were also taken on day 5 before liver biopsies were removed. In group (2), the mean value for SGPT increased significantly above the normal range on days 3 and 5, but SGOT, LDH, and LAP activities remained unchanged, indicating the absence of either liver injury or cholestasis. Peripheral fatty liver was present in 1/6 biopsies. This may have been due to failure of the dog to eat enough or to increase lipolysis induced by ethanol. In group (3), SGOT, SGPT and AP levels were significantly elevated on days 1, 3, and 5, indicating parenchymal liver damage with liver cell necrosis. Fatty liver was present in all dogs and may have been dietary in origin; two dogs had hepatic necrosis. The activities of all serum enzymes studied were considerably lower and liver biopsies showed fewer changes in dogs given high-calorie infusions + phosphate (groups four and five). Increased alkaline phosphatase activities in groups 2-5 were attributed to enzyme induction produced by the initial decrease in serum phosphate concentrations which, in turn, was caused by the high intake of carbohydrates and their metabolization. The hepatotoxic effect of solutions used for high-calorie infusion can be reduced by adding ATP or Sörensen buffer. The dose of phosphate administered is responsible for this effect.

6221 HORMONAL CONTRACEPTIVES AND LIVER DAMAGE. *(Ger.)* Otto, G.; Uhlig, K. (Polyclin., Dep. Minist., Berlin, Germany). *Z. Aerztl. Fortbild. (Jena)* 68(21):1181-1183, 1975.

Oral contraceptives have been prescribed for approximately 2000 women with normal histories, no evidence of hepatomegaly, and normal liver function tests. These patients were routinely followed every 6 months to determine SGOT, SGPT, and alkaline phosphatase activities. After taking oral contraceptives for 4-54 months, nine women, aged 20-37 yr, had abnormal liver function tests. A diagnosis of liver injury was confirmed by biopsy, intravenous cholangiography, or a liver specimen taken during cholecystectomy in all but two cases. The findings included diffuse fatty liver (one case), chronic persistent hepatitis (three cases), cholecystolithiasis (two cases), mild parenchymal damage (one case), cholangiolitis (one case), cholesteatosis of the gallbladder (one case), and Dubin-Johnson syndrome (one case). In four cases, patients were able to resume oral contraceptives after liver function tests had normalized. One patient stopped taking oral contraceptives, became pregnant and developed pruritis and increased alkaline phosphatase and SGPT values toward the end of pregnancy. Two months after delivery, oral contraceptives were resumed, but transaminase values increased and chronic persistent hepatitis was found on biopsy; oral contraceptives were discontinued. Oral contraceptives were also discontinued in one patient with cholangiohepatitis and one with Dubin-Johnson's syndrome and in two patients with no histological evidence of liver injury whose transaminase values increased again after a 1-month trial.

6222 PHENYLBUTAZONE AND HEPATITIS. *(E.)* Fowler, P. D.; Woolf, D.; Alexander, S. (Med. Dep., Geigy Pharm., Macclesfield, England). *Rheumatol. Rehabil.* 14(2):71-75, 1975.

A case is reported of a patient who developed mild hepatitis during a comparative trial of enteric-coated and rectally administered phenylbutazone in patients with rheumatoid disease. A 69-yr-old woman with rheumatoid arthritis was treated with gold sodium thiomalate, and received a total dose of 1040 mg gold. A course of phenylbutazone was begun 2 months after stopping the gold treatment and 1 month after stopping corticosteroids. Less than 1 month later, she was admitted with epigastric pain, nausea, vomiting and generalized fatigue. On examination she had vesicular lesions similar to those seen in chickenpox; the liver was slightly enlarged and tender and remained so for 21 days. Liver function tests were carried on on two occasions about 1 month later: eosinophilia was 1869 and 4544/mm^3, resp.; alkaline phosphatase and BSP retention were both elevated above normal limits for more than 1 month after the initial episode. Lymphocyte reactivity tests were carried out to determine the cause of jaundice, and phenylbutazone was identified as the probable causative agent. Close study of the relation of the jaundice to the drug therapy revealed that either phenylbutazone or gold sodium thiomalate malate could be incriminated, especially in view of the relative frequency of gold hepatitis and rarity of phenylbutazone hepatitis. This study indicates the difficulty of interpreting adverse reaction data, and the positive help from the lymphocyte transformation test. It is concluded that this patient should not be again exposed to phenylbutazone or related compounds, and that if gold is thought necessary, a very small test dose should first be given.

6223 JAUNDICE DUE TO ERYTHROMYCIN ESTOLATE. *(E.)* Lunzer, M. R.; Huang, S. N.; Ward, K. M.; Sherlock, S. (R. Free Hosp., London, England). *Gastroenterology* 68(5):1284-1291, 1975.

A case of jaundice and abdominal pain associated with erythromycin estolate therapy is reported. A 43-yr-old man received a short course of erythromycin estolate for acute pharyngitis; 1 yr later, he experienced another attack of pharyngitis, and was again placed on the drug (250 mg/8 hr). After 24 hr, he experienced severe epigastric pain requiring hospital admission. After 48 hr, his urine became dark, and he was found to develop jaundice; transaminase and bilirubin values were elevated, and viral hepatitis was considered. A further course of the antibiotic was given on another attack of pharyngitis, and the patient again experienced epigastric pain and jaundice. Electron micrographs

were taken of percutaneous liver biopsy before and after challenge with two doses of 250 mg erythromycin estolate. Pre-challenge, the general ultrastructures of the liver cells, bile canaliculi, and interlobular bile ducts were normal. A prominent degree of vesiculation was noted in periportal liver cells. About 20% of bile canaliculi, both in the centrilobular and periportal zones, exhibited a mild degree of dilatation. The portal areas showed inflammatory infiltration with presence of eosinophils immediately around the bile ducts. The results of liver function tests taken after challenge showed elevation of aspartate transaminase, alkaline phosphatase, and serum bilirubin. These findings indicate that many anatomical and cellular factors are involved in the mechanism of erythromycin estolate-induced liver injury, although the precise mechanism is not yet fully understood.

6224 GREY-SCALE ULTRASONOGRAPHY FOR MONITORING INDUSTRIAL EXPOSURE TO HEPATOTOXIC AGENTS. *(E.)* Taylor, K. J. W.; Williams, D. M. J.; Smith, P. M.; Duck, B. W. (R. Marsden Hosp., Sutton, England). *Lancet* 1(7918):1222-1224, 1975.

Preliminary results are reported demonstrating the use of grey-scale ultrasonography in the visualization of pathological lesions of the liver and spleen related to vinyl chloride exposure. Utilizing skin contact with a metallic transducer and a suitable coupling agent such as mineral oil, the liver was scanned in a series of parasagittal sections by moving the transducer through an arc. The resultant scan in a normal subject showed the normal liver consistency and, in a suitable parasagittal plane, a portal vein of normal size and appearance. In patients with portal hypertension, the portal vein was enlarged and tortuous. Although evidence of fatty infiltration and fibrosis was seen in these patients, the pathological appearance of the liver was much less obvious than that seen in patients with cirrhosis and an equivalent degree of portal hypertension. The spleen can be scanned along the 10th intercostal space with the patient lying in the right lateral position. A measure of spleen size can be obtained from the longitudinal axis. When splenomegaly is secondary to portal hypertension, it has a characteristic pattern which may be differentiated from malignant or chronic inflammatory causes of splenomegaly. These data suggest that grey-scale ultrasonography could be valuable for monitoring any liver or spleen changes related to vinyl chloride exposure. Attractive features of the ultrasound apparatus include the potential mobility in a vehicle of similar dimensions to a mobile chest x-ray unit, and the lack of ionizing radiation hazards and domestic power requirements. It is concluded that these features would permit the safe, non-invasive screening of whole populations exposed to potentially hepatotoxic agents.

6225 CARBON TETRACHLORIDE OVERDOSAGE: A CASE REPORT. *(E.)* Kennaugh, R. C. (Frere Hosp., East London, S. Afr.). *S. Afr. Med. J.* 49(15):635-636, 1975.

The successful management of liver and renal failure, ventricular fibrillation, hemorrhagic diathesis, and central nervous system agitation following carbon tetrachloride overdose in a 30-yr-old white male is described. The patient was hospitalized following the ingestion of a number of Carbrital capsules, trichloral, ethanol, and 120 ml of CCl_4. A stomach washout was performed 2 to 3 hr later. Initially 80 mg of furosemide was given i.v. every 8 hr as treatment for renal failure; after 1 day this was increased to 1 g every 12 hr and was stopped on day 5 when diuresis began and KCl was added to the daily i.v. infusion fluid. Treatment for hepatic failure included i.v. vitamin K (20 mg) administration on admission, followed by p.o. vitamin BCo and vitamin C administration and p.o. neomycin administration on day 5. I.v. verapamil (10 mg, twice daily) was given after Digoxin failed to control atrial fibrillation; spontaneous reversion to sinus rhythm occurred on day 22. I.v. (later oral) diazepam (10 mg every 6 hr) was necessary to control the patient's agitation and from day 4, i.v. Solu-Medrol (125 mg twice daily) was administered as a general treatment. The hemoglobin decreased slowly during hospitalization, the white count remaining high until discharge and the platelets falling gradually but increasing prior to discharge (29 days after admission). Most deaths following CCl_4 poisoning occur within 3 to 10 days as the result of massive liver necrosis or acute renal failure. If the toxic effects arising from CCl_4 poisoning are known and looked for, they can be treated successfully *ad initio*.

6226 HALOTHANE HEPATITIS. *(Fin.)* Rosenberg, P.; Yrjola, H. (Anesthesiol. Dept., Univ. Helsinki, Finland). *Duodecim* 91(3):29-38, 1975.

6227 CHOLESTATIC JAUNDICE AND PHENOBARBITAL. *(It.)* Buffoni, L.; De Santis, L. (Giannina Gaslini Inst., Genoa, Italy). *Minerva Pediatr.* 26(25):1278-1281, 1974.

6228 LIVER INJURY IN DRUG-DEPENDENT PATIENTS. *(Ger.)* May, B.; Helmstaedt, D.; Kramer, D.; Lotze, J.; Vido, I.; Schmidt, F. W. (Med. Coll., Hannover, Germany). *Verh. Dtsch. Ge Inn. Med.* 80:1554-1556, 1974.

6229 MEASUREMENT OF LIVER-CADMIUM CONCENTRATIONS IN PATIENTS AND INDUSTRIAL WORKERS BY NEUTRON-ACTIVATION ANALYSIS. *(E.)* Harvey, T. C.; Thomas, B. J.; McLellan, J. S.; Fremlin, J. H. (Dep. Med., Univ. Birmingham, England). *Lancet* (7919): 1269-1272, 1975.

6230 LYMPHOCYTE TRANSFORMATION IN HALOTHANE-RELATED HEPATITIS. *(E.)* Moult, P. J. A.; Adjukiewicz, A. B.; Gaylarde, P. M.; Sarkany, I.; Sherlock, S. (R. Free Hosp., London, England). *Br. Med. J.* 2(5962):69-70, 1975.

6231 ACETAMINOPHEN POISONING AND TOXICITY. *(E.)* Rumack, B. H.; Matthew, H. (Univ. Colorado Med. Cent., Denver). *Pediatr. Clin.* 55(6):871-876, 1975.

6232 HALOTHANE AND JAUNDICE. *(E.)* Simpson, B. R.; Strunin, L.; Walton, B. (London Hosp., England). *Br. J. Hosp. Med.* 13(4):433-436, 1975.

6233 ENCEPHALOPATHY AND FATTY INFILTRATION OF THE VISCERA (REYE-JOHNSON SYNDROME) A 17-YEAR EXPERIENCE. *(E.)* Chaves-Carballo, E.; Gomez, M. R.; Sharbrough, F. W. (Mayo Grad. Sch. Med., Rochester, Minn.). *Mayo Clin. Proc.* 50(4):209-215, 1975.

See also, 5382, 5440, 6003, 6239, 6247.

LIVER AND BILIARY TRACT

Acute Viral Hepatitis

6234 THE BALANCE OF TRACE ELEMENTS IN CHILDREN WITH INFECTIOUS HEPATITIS. *(Ukr.)* Trishkova, L. O. (Kiev Med. Inst., USSR). *Pediatr. Akush. Ginekol.* 35(2):16-18, 1973.

A balance study for trace elements in 20 children (nine boys and 11 girls, aged 6-11 yr) and in five healthy children was performed by spectroanalysis for copper, manganese, vanadium, nickel, and lead in food, feces, urine, and blood. The iron content was determined by the colorimetric Babenko method. It was observed that during illness, infected children receive in general fewer trace elements from their food than healthy children. During acute period of infection, or immediately after, iron, vanadium, nickel, lead, and copper leave the bodies of infected children at increased rates, through feces and urine, so that the balance of the trace elements in the bodies of children with infectious hepatitis is significantly offset, and requires additional supplement.

6235 SEROLOGIC CHARACTERIZATION OF PATIENTS WITH TWO EPISODES OF ACUTE VIRAL HEPATITIS. *(E.)* Karvountzis, G. G.; Mosley, J. W.; Redeker, A. G. (Univ. South. California Sch. Med., Los Angeles). *Am. J. Med.* 58(6):815-822, 1975.

Twenty-eight patients with two distinct episodes of illness compatible with acute viral hepatitis were reviewed to determine if presently available serologic techniques identified both episodes as attributable to hepatitis B virus, either because of recurrence or reinfection. No distinction was found between the first and second bouts with respect to clinical characteristics, abnormalities of liver function or epidemiologic background. Testing of serum specimens obtained during each of the two acute episodes, the interval between the two episodes and the period subsequent to the second, by sensitive procedures for hepatitis B surface antigens (HB_SAg) and their corresponding antibodies (anti-HB_SAg) permitted the following classification: 13 of 28 patients experienced first bouts serologically classifiable as due to hepatitis B virus; 11 had second bouts serologically classifiable as due to the hepatitis B virus; 2 patients had two episodes which were both serologically indeterminate; and 2 patients had two bouts neither of which appeared compatible with hepatitis B virus infection by present criteria. No patient had a second episode for which the HB_SAg and anti-HB_SAg data suggested hepatitis B virus recurrence or reinfection. These results do not favor speculation that the hepatitis B virus can account for repeated episodes of acute icteric hepatitis.

6236 RELATIONSHIP OF HEPATITIS B ANTIGEN IN CIRRHOSIS AND HEPATOMA IN THAILAND. AN ETIOLOGICAL SIGNIFICANCE. *(E.)* Chainuvati, T.; Viranuvatti, V.; Pongpipat, D. (Siriraj Hosp., Bangkok, Thailand). *Gastroenterology* 68(5):1261-1264, 1975.

A study was undertaken to determine the possible relation between hepatitis B surface antigen (HB_SAg), cirrhosis, and hepatoma. In a study of 221 patients, detectable HB_SAg was found in 16.3% of 49 patients who had hepatoma associated with cirrhosis. None of the 8 hepatoma patients without cirrhosis had detectable HB_SAg in the serum. When known causes of cirrhosis were excluded, HB_SAg was present in 18% of 22 patients. Positive α_1-fetoprotein (AFP) was found in 25 of 49 cases (51%) of hepatoma with cirrhosis but was found in only 1 of 8 cases (12.5%) of hepatoma without cirrhosis. Of 25 patients whose AFP was positive, HB_SAg was also present in 7. The latter was detected in only 1 of 24 patients in whom AFP was not detected. These results suggest that HB_SAg is closely associated with hepatomas in cirrhotic patients but not in noncirrhotic patients with hepatoma.

6237 THYMUS-DERIVED LYMPHOCYTES IN TYPE B ACUTE VIRAL HEPATITIS AND HEALTHY CARRIERS OF HEPATITIS B SURFACE ANTIGEN (HB_SAg). *(E.)* Wicks, R. C.; Kohler, P. H.; Singleton, J. W. (Univ. Colorado Med. Cent., Denver). *Am. J. Dig. Dis.* 20(6):518-522, 1975.

To assess thymus-derived (T) lymphocyte function in hepatitis B infection, lymphocytes from 8 patients with acute viral hepatitis type B and 8 healthy carriers of the hepatitis B surface antigen (HB_SAg) were compared with 13 normal controls using phyto-

hemagglutinin-induced blast cell transformation and sheep-cell rosette formation. The patients with acute hepatitis and the healthy carriers had serum HB_sAg detected by immunoelectrodiffusion within 1 week of *in vitro* lymphocyte testing. Overall T lymphocyte cell-mediated immune function was similar with the *in vitro* assays in all three groups, except for decreases in total T cells in 2 patients with acute disease. Consequently, if a defect in cellular immunity exists in normal controls with persistent hepatitis B infection, it is probably specific for the antigens of the hepatitis B virus and not related to a generalized impairment of T-lymphocyte function.

6238 FIBRINOGEN METABOLISM IN ACUTE HEPATITIS AND ACTIVE CHRONIC HEPATITIS. *(E.)* Clark, R. D.; Gazzard, B. G.; Lewis, M. L.; Flute, P. T.; Williams, R. (King's Coll. Hosp. Med. Sch., London, England). *Br. J. Haematol.* 30(1):95-102, 1975.

Coagulation studies, including those of radioactive fibrinogen metabolism, were performed in 7 patients with acute viral hepatitis and 12 with chronic active hepatitis. An increased fractional catabolic rate for fibrinogen was observed in 4 patients from the first group, and 7 from the second. In chronic active hepatitis, the catabolic rate was increased in those patients with the greatest degree of hepatic necrosis as demonstrated by raised serum aminotransferase levels. The correlation between the fibrinogen catabolic rate and extent of disease was less clear-cut in acute hepatitis, although the 2 patients with aspartate aminotransferase values >1000 IU/ml had the highest catabolic rates. There was no correlation between the rate of fibrinogen catabolism and the standard clotting tests, but the serial dilution of protamine sulfate test (indicating the presence of fibrin monomers in the circulation) was consistently positive in those cases where the catabolic rate was increased. These data, showing a correlation between an increased fractional catabolic rate and both a positive test for fibrin monomer and the plasma aminotransferase levels in active chronic hepatitis, suggest that this gradation in severity of intravascular coagulation may in some way be related to the severity of hepatic necrosis.

6239 ACUTE FULMINANT HEPATIC FAILURE: POTENTIAL THERAPEUTIC ROLE OF HEMOPERFUSION *(E.)* Willson, R. A. (Dep. Med., Univ. Washington, Seattle). *Gastroenterology* 69(1):244-248, 1975.

Hemoperfusion therapy techniques in the treatment of acute fulminant hepatic failure are discussed. The technique consists of perfusing whole blood through columns containing various toxin-removing adsorbants. Charcoal hemoperfusion uses activated charcoal coated with a thin biocompatible membrane to prevent loss of platelets and leukocytes in the charcoal, and to prevent escape of charcoal particles into the circulation. The technique is an extension of standard hemodialysis, and has a theoretically greater dialysance. Its effectiveness is dependent on whether the toxins are water-soluble, whether they will permeate the membrane, and whether they will be adsorbed by the charcoal (urea and ammonia will not). In studies already done, the technique was found to be simple, safe, and well tolerated by the patients. There was a 37% survival rate (of 37 patients with stage IV encephalopathy), as compared to a 13% survival rate (of 97 patients with stage III-IV encephalopathy) without hemoperfusion. Hemoperfusion using cation exchange resins will lower the blood ammonia concentration, but causes a decrease in circulating leukocytes and platelets. Encapsulating the resins with a biocompatible membrane is a probable solution. Anion exchange and uncharged resins have been shown to be superior to dialysis in removing protein-bound BSP, bilirubin, and bile acids; however, loss of leukocytes and platelets is significant. A suggested solution is to first separate the plasma by continuous centrifugation, or to develop more biocompatible resins. Albumin bound to agarose beads has been shown to remove protein-bound substances from blood with a minimal loss of leukocytes and platelets. It is concluded that hemoperfusion is a potentially useful technique in the treatment of acute liver failure.

6240 VIRAL HEPATITIS WITH INTRACYTOPLASMIC CHOLESTASIS AND CHOLESTEROL CRYSTALS IN A DRUG ADDICT. *(E.)* Livni, N.; Steigbuegel, D.; Levij, I. S. (Hebrew Univ. Hadassah Med. Sch., Jerusalem, Israel). *J. Pathol.* 116(1):47-50, 1975.

A case is presented of intracytoplasmic cholestasis and cholesterol crystals associated with viral hepatitis. A 16-yr-old female presented with jaundice, mild hepatosplenomegaly, and a temperature of 38 C, associated with upper abdominal pain, anorexia, nausea and vomiting. There was a history of drug abuse, and the patient was known to have used the syringe of a person with jaundice. There was an episode of similar symptoms three months prior to admission. Light microscopy of a liver biopsy showed a few acidophilic bodies and a minimal lymphocytic infiltrate compatible with a diagnosis of viral hepatitis. Thin sections revealed extensive intracellular bile pigment. Electron microscopy revealed focal degeneration of hepatocytes, masses of intracytoplasmic, osmiophilic, electron-dense material (presumably bile pigment), acidophilic round bodies (presumably necrotic cells), and round or oval virus-like particles about 500 to 800 Å in diameter with electron-light cores free in the cytoplasm. Also, numerous elongated, rhomboidal or rectangular empty clefts, 1 to 1.5 cm long, were found in the cytoplasm. They were surrounded by a layer of granular, electron-dense material and apparently represented localizations of cholesterol crystals dissolved during preparation of the tissue. Serum cholesterol level of the patient was normal. It is concluded that the intracellular precipitation of cholesterol crystals may have been due to an abnormal bile salt-cholesterol ratio related to intracellular cholestasis.

6241 HEPATITIS VACCINE: A NOTE OF CAUTION. *(E.)* Zuckerman, A. J. (no affil.). *Nature* 225(5504):104-105, 1975.

Current work toward the development of a safe and effective vaccine against viral hepatitis type B is reviewed. The ability of the isolated viral coat protein to challenge the body's immune mechanism in the same way as the whole infectious agent is known, and some research has developed a method for using purified 20 nm hepatitis B surface antigen particles which are free of detectable nucleic acid as immunogens. However, it is known that a variable amount of host protein and perhaps even carbohydrate may be complexed with hepatitis B viral protein in considerable quantities. If this is so, then these host proteins may include various pre-existing structures of the liver cell and may thus induce undesirable immunological reactions. There may be a similarity between the carbohydrate hapten, or the lipoprotein components found in purified fractions of the antigen, and those carbohydrate and lipoprotein antigens of normal cell surfaces, which would lead either to a degree of tolerance, or might initiate an autoimmune reaction. It is indicated that the immunological mechanisms and the presence of antibodies reacting with various tissue components may be involved in the pathogenesis of liver damage. Therefore, it may be undesirable to employ preparations of hepatitis B surface antigen which contain host cell components for immunization against this infection. Careful evaluation of immunization preparations in appropriate non-human primates is essential before small trials are undertaken in human subjects. These experimental trials should be directed not only at the protective efficacy of such immunogens but also should include careful assessment of all their effects on the immune system.

6242 THE RISK OF HEPATITIS FOLLOWING TRANSFUSION OF BLOOD POSITIVE FOR HEPATITIS B ANTIBODIES. *(Ger.)* Schlaak, M.; Lehmann, H.; Schober, H. (Med. Clin., Univ. Kiel, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:469-470, 1974.

Of 48 patients who underwent heart surgery with extracorporeal circulation, 25 developed hepatitis after incubation periods ranging from 28 to 110 days (mean 70 days); tests for hepatitis B antigen (HBAg) or antibody (HBAb) were positive in 90%. These patients received 3 to 42 U of blood (mean, 12.4 U/patient). There was a definite relation between the number of blood units received and the hepatitis risk in this group of patients. By using the maximum likelihood technique, the hepatitis risk was calculated at 50% with 10 U, and the risk/U was 6.4%. Radioimmunoassays showed that HbAg was present in only 1.1% of blood units administered to these patients, but HBAb was detected in 6%. In a larger group of 66 patients, eight had received at least 1 U of HBAg-positive blood and all eight developed hepatitis. Hepatitis occurred in 23 of these 66 patients, and in 14 of the 26 given HBAb-positive blood but in only nine of the 31 receiving HBAb-negative blood. However, ten patients given blood containing neither HBAg nor HBAb also developed hepatitis. By eliminating the use of blood containing either HBAg or HBAb, it is considered that the incidence of hepatitis could be reduced 70 to 80% in patients undergoing open-heart surgery.

6243 CHANGES IN IMMUNOGLOBULINS G, M, AND A AS ACUTE HEPATITIS EVOLVES TO CHRONIC HEPATITIS. *(Ger.)* Berthold, H.; Rotermund, H. M.: Maier, P.; Stiess, G.; Oehlert, W. (Hyg. Inst., Univ. Freiburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:474-476, 1974.

Quantitative immunoglobulin determinations were performed by radial immuno-diffusion on blood from patients with various forms of acute and chronic hepatitis to determine whether there is any relation between the humoral immune reaction in acute hepatitis and the further course of the disease. Patients consisted of 16 with uncomplicated acute hepatitis, six with acute hepatitis persisting for more than 4 months, four with chronic persistent hepatitis of more than 12 months duration, four with chronic aggressive hepatitis who recovered clinically and histologically without treatment, five with chronic aggressive hepatitis which had been observed to evolve from acute hepatitis, and seven with later stages of chronic aggressive hepatitis. Although the number of patients studied is too small to determine whether findings are statistically significant, there appeared to be a correlation between the primary humoral immune response and the course of acute hepatitis. Acute hepatitis in which the initial increase in IgM concentrations was small tended to evolve into the chronic persistent form, while patients with acute hepatitis accompanied by modest increases in immunoglobulin levels and a histological diagnosis of "chronic aggressive hepatitis" may recover spontaneously. Excessive increases in immunoglobulin levels at the onset of hepatitis suggest that evolution to chronic aggressive hepatitis may occur.

6244 SPONTANEOUS LYMPHOCYTE PROLIFERATION IN ACUTE AND CHRONIC HEPATITIS. *(Ger.)* Sodomann, C. P.; Rother, M.; Havemann, K. (Med. Clin., Univ. Marburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:1638-1641, 1974.

Lymphocyte proliferation in the absence of phytohemagglutinin (PHA) was studied in peripheral blood cultures from patients with Australia antigen (AuAg)-positive and AuAg-negative acute and chronic hepatitis by measuring ^{3}H-thymidine incorporation. Cultures contained 15% autologous serum or 15% pooled AB serum. Spontaneous lymphocyte proliferation occurred more often than in controls during the first 10 days after the onset of jaundice and before transaminase activities increased in patients with acute hepatitis B. This increase was more pronounced in the presence of homologous, rather than autologous, serum. The reason for this is not clear, but it may be due to the inhibition of increased DNA synthesis by AuAg-positive serum. In later stages of hepatitis B, values returned to the normal range. No difference in spontaneous lymphocyte proliferation was observed between early and later stages of hepatitis A, and there was little difference between results obtained in cultures containing autologous and those containing homologous serum. In all but a few patients with AuAg-positive chronic aggressive hepatitis, spontaneous lymphocyte proliferation was increased; the exceptions were patients with histologi-

cal evidence of cirrhosis. Values were in the normal range in patients with AuAg-negative chronic aggressive hepatitis and were below the normal range in patients developing cirrhosis. With one exception, lymphocyte proliferation was within normal limits in cultures obtained from patients with chronic persistent hepatitis, no matter whether AuAg was present or not. Spontaneous lymphocyte proliferation was significantly decreased in cultures from three patients with alcoholic cirrhosis and was greatly increased in cultures from three patients with infectious mononucleosis associated with hepatitis. These findings indicate that a large number of patients with AuAg-positive chronic aggressive hepatitis have lymphocytes which not only show increased reactivity to PHA, but also synthesize DNA at increased rates.

6245 IMMUNOGLOBULINS AND AUSTRALIN ANTIGEN IN VARIOUS FORMS OF HEPATITIS AND IN CIRRHOSIS. *(Ger.)* Reis, H. E.; Bertrams, J.; Selmair, H.; Wiese, W.; Kuwert, E.; Hirche, H. (Munic Hosp., Kassel, Germany). *Med. Welt.* 25(31-32):1277-1279, 1974.

Immunoglobulins were measured by radial immunodiffusion and hepatitis B antigen (HBAg) titers by the complement fixation reaction on blood from patients with various forms of acute and chronic hepatitis and cirrhosis to determine whether there is an inverse correlation between immunoglobulin levels and the appearance of HBAg. Blood was taken from 46 patients with acute and 25 with protracted hepatitis, 46 with chronic persistent and 58 with chronic aggressive hepatitis, 49 with cryptogenic and 18 with posthepatitic cirrhosis, and from 93 controls. Statistical analysis was performed logarithmically since, with the Kolmogoroff-Smirnoff test, immunoglobulin values are better described by a logarithmic, rather than a nonlogarithmic, normal distribution. Individual geometric means were compared by using the Duncan test. In chronic aggressive hepatitis and cryptogenic and posthepatitic cirrhosis, mean IgG and IgA values were significantly higher than in controls. IgG levels were also increased, but not to the same extent, in acute hepatitis. The greatest increases in IgM levels occurred in acute and protracted hepatitis, but significant increases were also apparent in chronic aggressive hepatitis and cryptogenic and posthepatitic cirrhosis; a smaller, but still significant, increase occurred in chronic persistent hepatitis. HBAg was detected in 43.4% of patients with acute hepatitis, 37.0% with protracted hepatitis, 72.6% with chronic persistent hepatitis, 48.0% with chronic aggressive hepatitis, and in 26.7% of the patients with both forms of cirrhosis. A comparison of immunoglobulin values in HBAg-positive blood with those in HBAg-negative blood showed no significant differences for any of the three immunoglobulins in any of the diseases considered. Similarly, no correlation was found between HBAg titers in chronic persistent and chronic aggressive hepatitis and the individual immunoglobulin levels. These findings conflict with others reported in the literature. Since no relation was observed between elimination of HBAg and immunoglobulin levels, increases in immunoglobulin levels occuring in various forms of hepatitis and cirrhosis must be induced by other antigens. It is suggested that the almost complete lack of immune response in chronic persistent hepatitis and reduced HBAg elimination in chronic aggressive hepatitis may be genetically determined, possibly by antigens in the histocompatibility system.

6246 HEPATITIS-B VIRUS AND INDIAN CHILDHOOD CIRRHOSIS. *(E.)* Nayak, N. C.; Roy, S.; Ramalingaswami, V.; Sachdeva, R. (All India Inst. Med. Sci., New Delhi). *Lancet* 2(7925):109-111, 1975.

Electron microscopy and immunospecific and orcein staining procedures were used to look for the components of hepatitis-B virus in the livers of children suffering from Indian childhood cirrhosis. The material consisted of 9 necropsy samples, 15 percutaneous needle biopsies, and 5 fresh-frozen liver biopsies. Immunostaining revealed neither the core nor surface component of hepatitis-B virus in any of the livers, and orcein staining from hepatitis-B antigen was also negative in all but one case. Coarse nonantigen granules, characteristic of degeneration hepatocytes, were often present, particularly in the necropsy material. Electron microscopic examined revealed no particles conforming in ultrastructural appearance to the hepatitis-B virus core antigen, although in a few cases occasional hepatocytes showed nonviral inclusions. The hepatocytic cytoplasm showed marked degenerative changes in the various organelles, but no proliferation of the smooth endoplasmic reticulum and no intracisternal structures resembling hepatitis-B surface antigen. These data, in conjunction with the other features of the disease, suggest that hepatitis-B virus infection plays no significant role in the etiology of Indian childhood cirrhosis.

6247 HEPATIC DYSFUNCTION IN RECIPIENTS OF RENAL ALLOGRAFTS. *(E.)* Berne, T. V.; Chatterjee, S. N.; Craig, J. R.; Payne, J. E. (Univ. Southern California Sch. Med., Los Angeles). *Surg. Gynecol. Obstet.* 141(2):171-175, 1975.

Findings of hepatic dysfunction in 18 of 42 recipients of renal allografts were analyzed with particular emphasis on the cause and results of altering the drug therapy. Following transplantation, immunosuppresive therapy consisted of prednisone and azathioprine. After an initial dose of 5 mg/kg of azathioprine, the patients received maintenance doses between 2.5 and 3.0 mg/kg/day. Prednisone was given at a dose of 2 mg/kg/day, tapered to 0.5 mg/kg/day by the end of the first month, and was reduced to 0.3 mg/kg/day by 6 months. Rejection was treated i.v. with methylprednisolone pulse therapy and the reinstitution of higher p.o. doses of prednisone. Hypertension frequently was treated with up to 2 g of methyldopa/day. Patients with thoracic roetgenographic evidence of primary pulmonary tuberculosis were given 300 mg of isoniazid/day. Evidence of a hepatic dysfunction was based on serum

transaminease levels greater than 200 IU or total serum bilirubin levels greater than 2 mg%. In ten patients, hepatic dysfunction occurred while the clinical course and renal function were otherwise stable. In this group, cyclophosphamide was substituted for azathioprine, and other potentially hepatotoxic drugs were discontinued. Indexes of hepatic function improved in all of these patients, and there was no deterioration of renal function. Of 11 patients with hepatic tissue available for histopathologic study, six demonstrated changes suggestive of viral hepatitis. Two had cholestasis, suggestive of drug injury, while in three others, changes were nonspecific. Only three of the 18 patients with hepatic dysfunction were persistently hepatitis B surface antigen-positive as measured by counterimmunoelectrophoresis. Cytomegalovirus was grown from the blood within 1 month after the first detection of abnormalities of the liver in five patients, and complement-fixing antibody titer rose in six of eight patients studied. All of the five patients with a histopathologic finding of viral hepatitis had evidence of cytomegalovirus infection, while none was positive for hepatitis B surface antigen. Although there is no clear cause for the hepatic dysfunction of renal allograft recipients, the results show that discontinuation of potentially hepatotoxic drugs is associated with improvement.

6248 ANTIBODY TO HEPATITIS-B CORE ANTIGEN IN PATIENTS WITH PRIMARY HEPATIC CARCINOMA. *(E.)* Maupas, P.; Larouze, B.; London, W. T.; Werner, B.; Millman, I.; O'Connell, A.; Blumberg, B. S.; Saimot, G.; Payet, M. (Fox Chase Cancer Cent., Philadelphia, Pa.). *Lancet* 2(7923):9-11, 1975.

Antibody to hepatitis-B core antigen (anti-HB_c) was assayed in the serum of 153 patients with primary hepatic carcinoma and in 238 controls from Hong Kong, West Africa, and the United States. All sera were tested by counter-electrophoresis and immunodiffusion against hepatitis-B core antigen (HB_cAg) extracted from the liver of a patient with chronic hepatitis. Sera which gave equivocal results were then tested by counter-electrophoresis against HB_cAg Amicon concentrate. Anti-HB_c was detected in 114 of the primary hepatic carcinoma patients, the percentage of positive patients being over 90% in Senegal and about 70% in Mali and Hong Kong. Of 21 American patients, only five were positive for anti-HB_c and one of these was Chinese while another was Puerto Rican. Among the controls, 20-68% of those from Asia and Africa were positive for anti-HB_c, while only 4% of those from the United States were positive. The data, in conjunction with previous findings, support the hypothesis that chronic infection with hepatitis-B virus is etiologically related to primary hepatic carcinoma, especially in Asia and Africa, although other factors must also be involved. With the development of a vaccine against the hepatitis-B virus, prevention of many cases of primary hepatic carcinoma may, therefore, be a realistic expectation.

6249 LYMPHOCYTE STIMULATION IN HEPATITIS B INFECTIONS. *(E.)* Tong, M. J.; Wallace, A. M.; Peters, R. L.; Reynolds, T. B. (Univ. Southern California Sch. Med., Los Angeles, Calif.). *N. Engl. J. Med.* 293(7):318-321, 1975.

Cell-mediated responses in patients with acute and chronic forms of Type B hepatitis were tested by lymphocyte transformation to purified hepatitis surface antigen (HB_sAg) and to phytohemagglutinin by dinitrochlorobenzene sensitization and by response to skin test antigens. Stimulation of human peripheral lymphocytes by purified HB_sAg or phytohemagglutinin was measured by uptake of ^3H-thymidine. During the acute phase of Type B hepatitis, lymphocyte transformation to HB_sAg was not detected in any patients. However, all acquired *in vitro* lymphocyte reactivity to HB_sAg during the early recovery period when SGPT levels and circulating HB_sAg titers were declining. Patients with Type B chronic active hepatitis and carriers of HB_sAg and patients with chronic persistent hepatitis uniformly showed no lymphocyte transformation to HB_sAg. Lymphocyte transformation to phytohemagglutinin was normal in all groups studied. Primary sensitization with topical application of 2000 µg of dinitrochlorobenzene was achieved in all six patients during the acute phase of Type B hepatitis. A delayed skin reaction to dinitrochlorobenzene developed in eight patients with Type B chronic active hepatitis and seven patients with chronic persistent hepatitis and HB_sAg carriers who were tested. The results support the hypothesis that the clinical course of hepatitis B virus infection depends on the competency of the thymus or T-dependent lymphocyte system. Recovery from acute Type B hepatitis is associated with the development of a specific cell-mediated response to purified HB_sAg during the early convalescent period.

6250 AGE DEPENDENCE OF THE OCCURENCE OF AUSTRALIA ANTIGENAEMIA IN PATIENTS WITH VIRAL HEPATITIS. *(Pol.)* Boron, P.; Sokolewicz, E.; Kossakowski, R. (Clin. Infectious Dis., Med. Acad., Bialystok, Poland). *Przegl. Lek.* 31(8):725-728, 1974.

6251 COMPARISON OF ULTRASTRUCTURAL AND IMMUNOHISTOCHEMICAL FEATURES OF HEPATOCYTES IN HBAg CARRIERS. *(It.)* Caramia, F.; Turbessi, G.; DeBac, C.; Ricci, G. (Inst. Gen. Pathol. II, Univ. Rome, Italy). *Boll. Ist. Sieroter. Milan* 53(3): 472-484, 1974.

6252 ABORTIVE INFECTIOUS HEPATITIS IN CHILDHOOD. *(Ger.)* Meinhard, F.; Herms, G. (Pediatr. Clin., Magdeburg-Altstadt Dist. Hosp., Germany). *Z. Gesamte Inn. Med.* 30(5):188-194, 1975.

6253 PREVALENCE OF HEPATITIS B ANTIGEN IN RESIDENTS AND HEPATITIS PATIENTS IN PERU. *(Sp.)* Ishida, N.; Umenay, T.; Miyamoto, T.; Mejia, J.; Flores, W.; Sanchez, S.; Mendez, R. (National Inst. Health, Univ. San Marcos, Lima, Peru). *Bol. Of. Sanit. Panam.* 78(5):445-451, 1975.

6254 ASSESSMENT OF PHAGOCYTIC ACTIVITY OF LEUCOCYTES IN VIRUS HEPATITIS. *(Pol.)* Kiczka, W.; Szkaradkiewicz, A. (Clin. Infect. Dis., Inst. Intern. Dis., Poznan, Poland). *Pol. Tyg. Lek.* 29(46): 1979-1982, 1974.

6255 FULMINANT VIRAL HEPATITIS. *(E.)* Williams, R. (no affiliation given). *Clin. Gastroenterol.* 3(2):419-436, 1974.

6256 VIRAL HEPATITIS: RECENT ADVANCES IN ITS PREVENTION. *(Fr.)* Vieira, J. (no affil.). *Recherche* 5(44):382-383, 1974.

6257 TREATMENT OF ACUTE VIRAL HEPATITIS. *(Ger.)* Franken, F. H. (St. Joseph Hosp., Wuppertal-Elberfeld, Germany). *Med. Welt* 25(46):1901-1904, 1974.

6258 ASPECTS OF THE ANAMNESIS AND CONCOMITANT DISEASES IN PATIENTS WITH VIRAL HEPATITIS. *(Rus.)* Demidova, A. N. (Dept. Infect. Dis., Ivano-Frankovsk Med. Inst., USSR). *Vrach. Delo* 616(2): 145-146, 1975.

6259 OZOKERITE THERAPY IN CHILDREN WITH VIRAL HEPATITIS. *(Rus.)* Rasnitsina, I. E.; Pisarev, A. G. (Dept. Infectious Infantile Dis., N. I. Pirogov Second Moscow Med. Inst., USSR). *Vopr. Okhr. Materin. Det.* 19(12):11-14, 1974.

6260 CLINICAL SIGNIFICANCE OF DETECTION OF THE AUSTRALIA ANTIGEN IN CHILDREN WITH VIRAL HEPATITIS. *(Rus.)* Petrunin, Y. P. (Dept. Infectious Infantile Dis., N. I. Pirogov Second Moscow Med. Inst., USSR). *Vopr. Okhr. Materin. Det.* 19(12):7-10, 1974.

6261 SOME PECULIARITIES OF THE CLINICAL PICTURE AND PATHOGENESIS OF INFECTIOUS AND SERUM HEPATITIS IN CHILDREN. *(Rus.)* Nisevich, N. I.; Bokova, A.G.; Petrunin, Y. P.; Cherednichenko, T. V.; Fedotov, V. V.; Nechaeva, T. A.; Pisarev, A. G. (Dept. Infectious Infantile Dis., N. I. Pirogov Second Moscow Med. Inst., USSR). *Vopr. Okhr. Materin. Det.* 19(12):3-6, 1974.

6262 EMPLOYMENT OF GAMMAGLOBULIN FOR THE PROPHYLAXIS OF RESIDUAL PHENOMENA AND COMPLICATIONS IN VIRAL HEPATITIS CONVALESCENTS. *(Rus.)* Latenko, Y. P.; Ziva, V. K. (Dept. Infectious Dis., N. I. Pirogov Vinnitsa Med. Inst., USSR). *Vrach. Delo.* 616(2):148-150, 1975.

6263 THE USE OF LAPAROSCOPY AND DIRECTED LIVER BIOPSY FOR THE STUDY AND DIFFERENTIAL DIAGNOSIS OF VIRAL HEPATITIS. *(Rus.)* Galperin, B. L.; Krichevskii, Iu. A.; Usviatsev, Ia. A. (Hosp. Infect. Diseases No 22, Kharkov, USSR). *Vrach. Delo* (4):117-119, 1975.

6264 MANIFESTATIONS OF THE HEPATO-RENAL SYNDROME IN VIRAL HEPATITIS. *(Rus.)* Nikiforov, V. N.; Latsinik, G. E. (Dept. Infectious Dis., Central Inst. Postgrad. Training Phys., Moscow, USSR). *Sov. Med.* (11):89-93, 1974.

6265 VIRAL HEPATITIS AND CIRRHOSIS. *(E.)* Bjorneboe, M. (no affiliation given). *Clin. Gastroenterol.* 3(2):409-418, 1974.

6266 ETIOLOGICAL SPECTRUM OF POST-TRANSFUSION HEPATITIS. *(E.)* Knodell, R. G.; Bell, C. J.; Conrad, M. E. (Dep. Hematol., Walter Reed Army Inst. Res., Washington, D.C.). *Gastroenterology* 68(4):927, 1975.

6267 HYPOGLYCEMIA AND AGLYCEMIA IN VIRAL HEPATITIS. *(Ger.)* Lindner, W. (Munic, Hosp., Salzgitter-Lebenstedt, Germany). *Munich. Med. Wochenschr.* 117(20):855-860, 1975.

6268 THE FRUCTOSE-1-PHOSPHATE ALDOLASE IN THE DIAGNOSIS OF AND PROGNOSIS FOR VIRAL HEPATITIS IN CHILDREN. *(Rus.)* Balabonkin, V. G.; Milman, M. Sh.; Uzbekova, Kh. Z. (Uzbek. Res. Inst. Pediatr., Tashkent, USSR). *Vopr. Okh. Materin. Det.* 19(11):85, 1974.

6269 THE DIAGNOSIS AND TREATMENT OF CHOLESTATIC FORMS OF VIRAL HEPATITIS. *(Fr.)* Vachon, A. (Hotel Dieu, Lyon, France). *Cah. Med.* 15(15):1071-1077, 1974.

6270 DEVELOPMENT DISORDERS CAUSED BY VIRAL HEPATITIS IN YOUNG PATIENTS. *(Fr.)* Laverdant, C.; Molinie, C. (Begin Hosp., St. Mande, France). *Cah. Med.* 15(15):1049-1063, 1974.

6271 THE SUBTYPES OF HEPATITIS B ANTIGEN (AUSTRALIA ANTIGEN). *(Ger.)* Frank, K. H.; Reimann, W.; Renger, F. (Carl Gustav Carus Med. Acad., Dresden, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):245-247, 1974.

6272 THE DANGEROUS INCREASE IN ACUTE AND CHRONIC LIVER DISEASE. *(Ger.)* Muting, D. (no affil.). *Fortschr. Med.* 93(8):393-394, 1975.

6273 DISTRIBUTION PATTERN OF AUSTRALIA ANTIGEN SUBTYPES D AND Y IN LIVER DISEASE. *(Ger.)* Schoppe, W. D.; Kindler, V. (Med. Clin., Univ. Dusseldorf, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80: 463-465, 1974.

6274 COMPARISON OF LABORATORY FINDINGS IN ACUTE HB-Ag-POSITIVE AND HB-Ag-NEGATIVE HEPATITIS. *(Ger.)* Linhart, P.; Kommerell, B.; Erbe, R.; Schneid, M. (Med. Clin., Univ. Heidelberg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:471-472, 1974.

6275 THE CURRENT EPIDEMIOLOGICAL STATUS OF VIRAL HEPATITIS IN THE HANNOVER AREA. *(Ger.)* Wehrmann, M.; Muller, R.; Kramer, R.; Willers, H.; Knocke, K. W.; Deicher, H.; Hopken, W. (Med. Coll., Hannover, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:510-513, 1974.

6276 FLUORESCENCE SEROLOGICAL DETECTION OF HEPATITIS B-ASSOCIATED ANTIGEN (Au Ag) IN PERIPHERAL BLOOD CELLS: FOLLOW-UP STUDIES IN ACUTE HEPATITIS AND CONTROLS WITH NORMAL LIVERS. *(Ger.)* Arnold, W.; Volker, K.; Meyer zum Buschenfelde, K. H. (Med. Clin., Univ. Mainz, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:461-463, 1974.

6277 CLINICAL PROBLEMS OF THE AUSTRALIA ANTIGEN (HEPATITIS-B-ANTIGEN). *(Ger.)* Kaboth, V. (Clin., Univ. Gottingen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:344-349, 1974.

6278 THE CLINICAL VALUE OF DETERMINING HEPATITIS B ANTIBODIES. *(Ger.)* Lehmann, H.; Schober, A.; Schlaak, M. (Med. Clin., Univ. Kiel., Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:456-457, 1974.

6279 REPLICATION OF HEPATITIS B VIRUS IN CELL CULTURES. *(Fr.)* Panouse-Perrin, J.; Courouce-Pauty, A. M. (no affil.). *Ann. Immunol. (Paris)* 126 C(1):100-101, 1975.

6280 EPIDEMIOLOGY AND PREVENTION OF VIRAL HEPATITIS. *(Fr.)* Cappel, R. (no affil.). *Brux. Med.* 55(1):17-22, 1975.

6281 SERUM ENZYME ACTIVITY IN PATIENTS OF DIFFERENT AGE GROUPS WITH VIRAL HEPATITIS. *(Rus.)* Botsvadze, E. S.; Katsitadze, T. K.; Tavartkiladze, D. A. (Tbilisi Med. Inst., USSR). *Lab. Delo* (12):720-722, 1974.

6282 CLINICAL EVALUATION OF ENZYME UV-TESTS AND BIO-LA-TESTS USED IN COMBINATION WITH ULTRAMICROSYSTEM FOR EXAMINATION OF PATIENTS WITH VIRAL HEPATITIS. *(Rus.)* Luchshev, V. I.; Lezhnev, I. A. (Moscow 2nd Med. Inst., USSR). *Lab. Delo* (3):144-145, 1975.

6283 METABOLISM OF SOME AMINO ACIDS AND UREA IN THE BLOOD SERUM IN INFECTIOUS HEPATITIS. *(Rus.)* Khochava, A. I.; Vasiliev, V. S. (Grodno Med. Inst., USSR). *Lab. Delo.* (3):142-143, 1975.

6284 CHANGES OF SOME INDICATORS OF NONSPECIFIC IMMUNOLOGICAL REACTIVITY IN CHILDREN WITH VIRAL HEPATITIS. *(Ukr.)* Zakrevskii, N. N. (Kharkov Med. Inst., USSR). *Pediatr. Akush. Ginekol.* 37(2): 27-29, 1975.

6285 ACUTE SPORADIC HEPATITIS IN THE ADULT: CLINICAL, ETIOLOGICAL AND PROGNOSTIC CONSIDERATIONS ON FOLLOW-UP CASES WITH TYPICAL AND ATYPICAL SYMPTOMS. *(It.)* Coltorti, M.; Del Vecchio-Blanco, C.; Caporaso, N. (Fac. I. Med. Chir., Univ. Naples, Italy). *Recent Prog. Med. (Roma)* 56(3):238-274, 1974.

6286 TRANSPORT OF THE HEPATITIS B ANTIGEN. IMMUNOFLUORESCENCE STUDIES ON THE LOCATION OF THE ANTIGEN IN THE LIVER. *(It.)* Turbessi, G.; De Bac, C.; Caramia, F.; Ricci, G. (Inst. Infect. Dis., Univ. Rome, Italy). *Recent. Prog. Med. (Roma)* 56(3):212-229, 1974.

6287 INDEPENDENT EXTRAHEPATIC MANIFESTATIONS OF THE DISEASED LIVER IN VIRAL HEPATITIS. *(Fr.)* Paris, J. (no affil.). *Lille Med.* 19(10):1015-1034, 1974.

6288 KINETICS OF HB_S ANTIGEN IN MAN. *(E.)* Drouet, J.; Courouce-Pauty, A. M.; Thevenoux, A. M.; Soulier, J. P.; Chanard, J.; Vallee, G.; Funck-Brentano, J. L. (Necker Hosp., Paris, France). *Biomedicine* 22(2):158-166, 1975.

See also, 5466, 6143, 6506.

LIVER AND BILIARY TRACT

Chronic Hepatitis

6289 LONG-TERM IMMUNOSUPPRESSIVE THERAPY IN CHRONIC AGGRESSIVE HEPATITIS AND CIRRHOSIS. *(Ger.)* Bannaski, H. (Kempfenhausen Priv. Clin., Germany). *Med. Welt* 25(39):1551-1555, 1974.

Changes in histological findings, transaminase values, immunoglobulins, and clinical findings were compared in 212 patients with chronic aggressive hepatitis or compensated or decompensated cirrhosis who received immunosuppressive agents and in 88 patients who received conservative therapy (bed rest, diet, and various infusions and anabolic and hepatotropic agents) for 3 months to 10 yr. Of these patients, 52% were men and 48% were women; 69% were more than 50 yr old and only six were younger than 30 yr. Immunosuppressives were given to patients

with autoimmune diseases, primary biliary cirrhosis, and to patients with negative immunological findings who failed to respond to conservative therapy. Most patients in this group received cyclophosphamide or azathioprin, but some were given D-penicillamine and/or corticosteroids. A combination of azathioprin and corticosteroids is particularly recommended for cirrhosis. Initial doses of azathioprin were 50-150 mg, combined with 4-24 mg of corticosteroids. Corticosteroid dosages were then reduced as rapidly as possible to prevent serious side effects. Among patients given immunosuppressives, transaminase values improved in 90%, immunoglobulin G values improved in 80%, and histological findings improved in almost 70%. No comparable values were reported for patients receiving conservative therapy. Improvements occurred in 71% of 36 patients with compensated cirrhosis who received immunosuppressives and in 67% of 21 patients with decompensated cirrhosis. Leukopenia and thrombopenia were observed in 28% of patients given immunosuppressives, but were reversible when drug therapy was discontinued. Septicemia, mycosis, complete alopecia and gastric bleeding were observed in one patient each. Of eight patients with primary biliary cirrhosis, two died of cerebrovascular accidents, which were considered to have resulted from extremely high cholesterol levels after 7 and 9 yr of immunosuppressive therapy, resp.

6290 COMPLEMENT DETERMINATIONS IN LIVER DISEASE. *(Ger.)* Opferkuch, W.; Berger, J.; Forster, E.; Hopf, U.; Meyer zum Buschenfelde, K. H. (Dep. Med. Microbiol., Ruhr Univ. Bochum, Germany). *Z. Gastroenterol.* 13(suppl.):282-284, 1975.

Determinations of the fourth component of complement (C_4) were performed on 79 patients with histologically confirmed chronic active hepatitis, and the values were compared with those previously measured on normal controls. Since the mean C_4 concentration was only slightly decreased in chronic active hepatitis, the concentrations obtained were plotted against the percentage of patients having each concentration to obtain a distribution curve. At the maximum for the control curve, the curve for patients with hepatitis had a shoulder, suggesting that this group of patients contained at least two subgroups. Curves were then plotted for Australia antigen (AuAg)-positive and AuAg-negative patients. While the curve for AuAg-positive patients followed the normal distribution, C_4 concentrations were decreased in AuAg-negative patients, but the latter group also included patients with normal C_4 concentrations. Since C_4 and the second component of complement (C_2) are both produced in cells of the mononuclear, phagocytic system and since a similar decrease did not occur in C_2 concentrations in AuAg-negative patients with chronic active hepatitis, it is concluded that decreased C_4 concentrations in these patients are not caused by a disorder in the synthesis of complement.

6291 CHRONIC AGGRESSIVE HEPATITIS IN CHILDHOOD. *(Ger.)* Scholz, H. (Munic. Clin., Berlin-Buch, Germany). *Kinderaerztl. Prax.* 42(11): 487-494, 1974.

In a 7-yr period, seven children (five girls and two boys, aged six months to 9.2 yr at onset) were treated for chronic aggressive hepatitis. These patients were followed for 1.7 to 7.2 yr (mean, 5.0 yr). At onset, two children had acute viral hepatitis; two others, who were completely asymptomatic, were suspected of having subclinical hepatitis because of close contact with a hepatitis patient. The other three patients were suspected of recurrences of chronic hepatitis. Hepatomegaly was present in 4 cases and splenomegaly in only one. Increased SGPT activities were the most useful laboratory test in diagnosing recurrences. Australia antigen was detected in five of these patients; the first test for this antigen was made only 4 yr after onset in the two patients with negative findings. Signs of aggressive hepatitis were found in only one of the 12 to 15 biopsies performed on three children. One patient had cirrhosis on laparoscopy, but not on biopsy. Azathioprin alone, given to three patients, produced no improvement so it was combined with prednisolone. Two of these patients gradually responded after 6 months and 1 yr, resp., but no effect was noted in the remaining child and he had a severe recurrence lasting many months. All children received prednisolone, which appeared to give better results than azathioprin, but no convincing evidence was obtained that it had any long-term effect on progression of the disease and some serious side effects occurred (steroid diabetes, bone changes, infections).

6292 INCREASED DNA SYNTHESIS IN LYMPHOCYTES IN CHRONIC ACTIVE HEPATITIS. *(Ger.)* Muerker-Alzer, G.; Schumacher, K.; Gross, R. (Med. Clin., Univ. Cologne-Lindethal, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:1636-1638, 1974.

DNA synthesis was studied by measuring ^{14}C-thymidine incorporation in the absence of phytohemagglutinin (PHA) in 48-hr peripheral lymphocyte cultures from 76 patients with chronic aggressive hepatitis or active cirrhosis. In cultures from 24 of these 76 patients the rate of DNA synthesis was significantly faster than that in cultures from 48 controls. No correlation was found between this increase in DNA synthesis and any of the immunological parameters studied (immunoglobulins; antinuclear, antiglomerular, or antivascular antibodies; or hepatitis B antigen or antibody). PHA stimulation was not reduced in patients with *in vivo*-activated lymphocytes. In follow-up examinations, performed on 12 patients with *in vivo*-activated lymphocytes, clinical improvements, which occurred in four cases, were accompanied by normalization of resting DNA synthesis. No appreciable change occurred in lymphocytes from the remaining eight patients who died in hepatic coma during the observation period. *In vivo*-activated lymphocytes were large cells with round- or bean-shaped nuclei and resembled blast cells after PHA stimulation. Since these lymphocytes did not form rosettes spontaneously with sheep RBC, it is likely that they were B-lymphocytes. *In vivo*-activated lymphocytes are considered to be a manifestation of an immune reaction occurring in chronic aggressive hepatitis.

6293 TREATMENT OF CHRONIC AGGRESSIVE HEPATITIS WITH D-PENICILLAMINE. *(Ger.)* Heckers, H.; Gerhardt, H.; Leinweber, B.; Matthes, K. J. (Cent. Intern. Med., Univ. Gottingen, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:506-510, 1974.

D-penicillamine was used in the treatment of 32 patients, most of whom were hospitalized with chronic aggressive hepatitis and had been treated unsuccessfully with other drugs. Starting with 900 mg, the dosage was increased by 300 mg every 3 days so that a maximum of 1800 mg/day was reached in 13 days. This drug was combined with 300 mg/day of vitamin B_6 and was administered for as long as the blood picture permitted. Evaluation of histological findings in 14 patients who had been treated for as long as 20 months (mean 14 months) showed improvement in nine, no change in three, and deterioration in two. Of the latter two patients, one had cirrhosis and the other had a very active, chronic aggressive hepatitis which evolved into complete cirrhosis with portal decompensation within 4 months. At biopsy, three patients showed chronic persistent hepatitis and two had only residual periportal infiltrates; these two patients had been treated unsuccessfully for 4 and 2 yr, resp., with 75 to 100 mg/day of azathioprin, at times in combination with prednisolone. One seriously ill patient developed agranulocytosis in a 3-day period, but had a remission after D-penicillamine was discontinued and intensive antibiotic therapy was administered for 14 days. Another patient developed threatening leukopenia after 6 months of therapy, but was able to continue D-penicillamine when the dose was reduced to 600 mg/day. Some patients noted reversible changes in their sense of taste and others temporarily lost it. These disorders were traced by electrogustometry to areas innervated by the glosso-pharyngeal nerve; areas innervated by the facial nerve remained unaffected. Trials of copper salt substitution are planned for these patients. The results indicate the relatively high efficiency of D-pencillamine, and that intermittent leukopenia and granulocytopenia are not an absolute counterindication for this therapy.

6294 DIAGNOSTIC AND THERAPEUTIC ASPECTS OF CHRONIC AGGRESSIVE HEPATITIS. *(Ger.)* Gronauer, G.; Mauersberger, H.; zu Dohna, P. (Villingen-Schwenningen Munic. Hosp., Germany). *Therapiewoche* 24(37):4001-4002, 1974.

In 12 patients with chronic aggressive hepatitis, confirmed by laparoscopy or liver biopsy, quantitative determination of immunoglobulins was useful in routine diagnosis. IgG values were generally increased the most, with smaller increases in IgA and IgM. If IgG concentrations were normal, only IgM concentrations were increased. Patients with antinuclear and anti-smooth-muscle antibodies were given azathioprin + prednisone (doses unspecified), while those in whom no organ-specific antibodies were detected received D-penicillamine and vitamin B_6 (doses unspecified). Patients were then followed for six months to six yr. Almost all of these patients had been unsuccessfully treated with prednisone alone. Distinct improvement or normalization of immunoglobulin fractions, transaminase activites, and liver size and consistency occurred in all cases. Clinical and laboratory findings normalized completely in one patient given azathioprin + prednisone, and no recurrence developed in a two-yr period. The results of treatment were better and improvement occurred more rapidly in patients given D-penicillamine and vitamin B_6. Two patients who were initially treated with azathioprin + prednisone and responded favorably had recurrences of hepatitis when they were given D-penicillamine and vitamin B_6 instead. Distinct improvement occurred a short time after these patients were again treated with azathioprin + prednisone. Treatment with D-penicillamine and vitamin B_6 was unsuccessful in a patient with chronic aggressive hepatitis and organ-specific antibodies. Normalization of transaminase values and in the liver size and consistency occurred shortly after a change to azathioprin and prednisone. Therapy was consistently successful in the group of patients with chronic aggressive hepatitis and no organ-specific antibodies who were treated first with D-penicillamine and vitamin B_6.

6295 HEPATITIS-B ANTIGENAEMIA ASSOCIATED WITH ACTIVE CHRONIC HEPATITIS AND MESANGIOPROLIFERATIVE GLOMERULONEPHRITIS. *(E.)* Stratta, P.; Camussi, G.; Ragni, R.; Vercellone, A. (Cattedra Nefol. Med., Univ. Turin, Italy). *Lancet* 2(7926): 179, 1975.

The occurrence of hepatitis-B antigenemia in a 33-yr-old patient with active chronic hepatitis-associated mesangioproliferative glomerulonephritis is reported. The patient was hospitalized with a 1-month history of increasing weakness, peripheral edema, and dark urine. His serum contained hepatitis-B surface antigen but not antibodies. Active chronic hepatitis was diagnosed after examination of a liver biopsy specimen. Microhematuria, cylindruria, and unselective proteinuria were also found; and immunoglobulin G (IgG) was increased while serum-complement (C3 and C4) levels were below normal. Glomerular filtration was 70 ml/min and urinary fibrin-degeneration products were seen. Light and electron microscopic examination of a percutaneous renal biopsy specimen showed mesangial deposits and proliferation; immunohistochemistry revealed segmentary subendothelial deposits of IgG and immunoglobulin M widespread and generalized mesangial immunoglobulin A deposits, positive segmentary subendothelial immunofluorescence of lqC and C4 complement deposits, and diffuse and generalized subendothelial mesangial deposits of C3. There was hepatitis-B antigen in the glomerular mesangium. The evidence suggested the involvement of a circulating immune complex in the pathogenesis of the disease. It is concluded that serum and glomerular hepatitis-B antigen should be sought in glomerulonephritis of all histological types.

6296 ELECTROPHORETIC INTRA-CUTANEOUS INTRODUCTION OF MAGNESIUM IN THE COMBINED TREATMENT OF CHRONIC LIVER DISEASE IN CHILDREN. *(Rus.)* Ananeva, K. A.; Savelieva, L. A.; Nikolaeva, V. V.; Dedikova, L. A. (M. F. Vladimirskii Reg. Res. Inst. Clin. Med., Moscow, USSR). *Vopr. Okhr. Materin. Det.* 20(3):14-15, 1975.

6297 HEPATITIS AND CIRRHOSIS. *(Ger.)* Thaler, H. (Wilhelmine Hosp., Vienna, Austria). *Dtsch. Med. Wochenschr.* 100(18):1018-1025, 1975.

6298 THE IMMUNE PATHOGENESIS AND IMMUNODIAGNOSIS OF CHRONIC INFLAMMATORY LIVER DISEASES. *(Ger.)* Warnatz, H. (Univ. Hosp., Erlangen, Germany). *Med. Welt* 25(40):1592-1598, 1974.

6299 IMMUNE PROPHYLAXIS AND IMMUNOSUPPRESSION IN CHRONIC INFLAMMATORY LIVER DISEASE. *(Ger.)* Scheiffarth, F. (Univ. Hosp., Erlangen, Germany). *Med. Welt* 25(40):1589-1592, 1974.

6300 TREATMENT OF CHRONIC HEPATITIS AND CIRRHOSIS. *(Ger.)* Kommerell, B. (Med. Clin., Univ. Heidelberg, Germany). *Med. Welt* 25(46):1904-1910, 1974.

6301 DISTURBANCE OF LIPID METABOLISM IN PATIENTS WITH CHRONIC HEPATITIS AND LIVER CIRRHOSIS. *(Rus.)* Kurdybailo, F. V.; Mekhanik, Z. I. (Leningrad Sanit. Hyg. Med. Inst., USSR). *Klin. Med.* 53(1):64-67, 1975.

6302 TREATMENT OF CHRONIC HEPATITIS. *(Fr.)* Dioguardi, N. (Inst. Med. Pathol., Univ. Milan, Italy). *Cah. Med.* 15(10):663-667, 1974.

6303 CHRONIC VIRAL HEPATITIS. *(Fr.)* Darnis, F. (St. Antoine Hosp., Paris, France). *Cah. Med.* 15(15):1065-1070, 1974.

6304 THE DEVELOPMENT OF HEPATOMAS IN TWO PATIENTS WITH CHRONIC HEPATITIS AND HEPATITIS-ASSOCIATED ANTIGEN. *(Sp.)* Sotto, A.; Jimenez, G.; Ferret, O.; Oliva, J. (Inst. Gastroenterol, Havana, Cuba). *Rev. Esp. Enferm. Apar. Dig.* 45(3):317-324, 1975.

6305 CHRONIC HEPATITIS: NEW CLASSIFICATION AND CLINICAL CRITERIA. *(It.)* Del Prete, S.; Malacco, E.; Ucelli, S.; Doniach, D.; Dane, D. S.; Walsh, J. H. (Inst. Clin. Med. II, Univ. Milan, Italy). *Recent. Prog. Med. (Roma)* 56(3):193-211, 1974.

6306 CHANGES IN AUSTRALIA ANTIGEN (HB Ag) IN CHRONIC HEPATITIS BEFORE AND AFTER CORTICOSTEROID TREATMENT. *(It.)* Migneco, G.; Tripi, S.; Didato, A. (Inst. Gen. Clin. Med. Med. Ther., Univ. Palermo, Italy). *Boll. Soc. Ital. Biol. Sper.* 50(13):976-979, 1974.

6307 DIAGNOSIS AND THERAPY OF CHRONIC HEPATITIS. *(It.)* Messini, M. (Sch. Spec. Liver Dis., Univ. Rome, Italy). *Clin. Ter.* 71(6):505-520, 1974.

6308 THE EFFICACY OF TREATING PATIENTS WITH CHRONIC HEPATITIS AND HEPATOCHOLECYSTITIS BY MINERAL WATER AT ANKAVAN HEALTH RESORT. *(Rus.)* Ambartsumian, M. S.; Oganesian, D. A. (Ankavan Sanatorium, Armenia, USSR). *Vopr. Kurortol. Fizioter. Lech. Fiz. Kult.* (1):89-90, 1975.

6309 $ALPHA_1$-FETOPROTEIN IN CHRONIC HEPATITIS. *(It.)* De Benedetti, F.; Piombo, I. (Munic. Hosp., Genoa, Italy). *Minerva Gastroenterol.* 20(2):76, 1974.

6310 SERUM ARYLESTERASE ISOENZYMES IN CHRONIC HEPATITIS. *(E.)* Burlina, A.; Galzigna, L. (Gen. Hosp., Conegliano Veneto, Italy). *Clin. Biochem.* 7(3):202-205, 1975.

See also, 6097, 6101, 6127, 6168, 6187, 6238, 6243, 6244, 6245, 6249, 6272, 6273, 6277, 6338, 6523.

LIVER AND BILIARY TRACT

Alcoholic Liver Disease

6311 CONTRIBUTION TO ALCOHOLIC HEPATITIS. *(Ger.)* Stotzner, H. (Carl Gustav Carus Med. Acad., Dresden, Germany). *Zentrabl. Allg. Pathol.* 119(1/2):27-32, 1975.

Microscope examinations were performed on frozen and paraffin-embedded sections of punch liver biopsies taken from 64 patients (54 men and ten women, aged 26-69 yr) who had histories of drinking alcohol (mean 86 g/day) for long periods (mean 9.6 ± 5.2 yr) but who had no evidence of biliary disease or right heart failure or a history of exposure to hepatotoxic agents. Of these 64 patients, 30 (22 men and eight women) had alcoholic hepatitis. This was defined as neutrophilic granulocytes constituting: (1) about 1/3 of inflammatory cells in at least one affected portal field, (2) about 1/3 of inflammatory cells in one large intralobular focus of inflammation and (3) at least 1/2 of the cells in three or more small foci of inflammation. Hepatocytic necrosis was found in all 64 biopsies and the extent of this necrosis was not appreciably different in patients with alcoholic hepatitis and those without it. Of the 30 biopsies from patients with alcoholic hepatitis, all displayed steatosis; 15 had hydropic swelling, 19 had alcoholic hyalin, 17 had edema of the

portal fields and 21 had bile duct proliferation. Two of these histological features were identified in seven patients; three were seen in 11 patients, four were seen in six patients and five were seen in six patients. No significant difference was found between the daily alcohol consumption or the duration of alcohol ingestion in patients with alcoholic hepatitis and those without it.

6312 IMMUNOLOGIC REACTIVITY AND ALCOHOLIC LIVER DISEASE. *(E.)* Zetterman, R. K.; Leevy, C. M. (Coll. Med. Dent. New Jersey, Newark). *Bull. NY Acad. Med.* 51(4):533-544, 1975.

The investigation of cell-mediated immunologic reactivity by means of lymphocyte transformation and the production of migration-inhibition-factor (MIF) improves the diagnosis and therapeutic perspective in the alcoholic with liver disease. Reduced lymphocyte reactivity to the mitogen phytohemagglutin (PHA) is frequently observed in malnourished alcoholics with liver disease. This can be attributed to a reversible disease in the synthetic capacity of DNA which occurs as a result of liver injury, nutritional deficiency, or circulating antagonists. It is suggested that immunologic hyperreactivity to alcoholic hyaline may be of crucial importance in the transformation of alcoholic hepatitis to cirrhosis. In spite of a decrease in the total number of T cells and reduced responsiveness to PHA, alcoholic hyaline evokes an increase in migration inhibition factor. Supernatants of sensitized lymphocytes, stimulated with this material, produce an increase in the incorporation of proline into collagen. It is suggested that this actin-like protein may serve as a neoantigen and may thus stimulate antibody-forming cells of T lymphocytes to release biologically active products. Immunologic competence, reflected in lymphocyte response to PHA, increases in the alcoholic, with improvement of hepatic reserve induced by abstinence and correction of nutritional deficiencies. Immunologic hyperactivity with release of migration-inhibition factor and fibrogenic factor persists despite progressive deterioration of the liver and eventually leads to a vicious cycle. It is felt that drugs which suppress immunologic response and reduce fibrogenesis may be useful therapeutic adjuncts in selected alcoholics with progressive hepatic-cell destruction despite abstinence from alcohol and a good diet.

6313 ABNORMAL RENAL ACIDIFICATION IN ALCOHOLIC LIVER DISEASE. *(E.)* Oster, J. R.; Hotchkiss, J. L.; Carbon, M.; Vaamonde, C. A. (Miami VA Hosp., Fla.). *J. Lab. Clin. Med.* 85(6):987-1000, 1975.

Studies were undertaken to better define the incidence and characteristics of acidification abnormalities in alcohol-induced liver disease and to demonstrate the practicality of using a calcium chloride ($CaCl_2$) acid-loading test of short duration in patients in whom ammonium chloride (NH_4Cl) might precipitate hepatic encephalopathy. After ingestion of 150 mEq $CaCl_2$, urinary acidification was studied for 6 hr in 22 normokalemic patients with alcoholic liver disease of varying severity and in 7 control subjects during 10 studies. The degree of the induced acidosis was similar in both groups. Of the liver disease patients, 9 were unable to normally lower urine pH below 5.25 and these were compared with the 13 patients achieving lower pH and with controls. This defect was consistently produced. Titratable acid excretion was less in the 9 patients than in the other groups. The percentage contribution of ammonium to maximal net acid excretion was significantly higher in all patients with liver disease than in controls. None of the group of 9 patients had spontaneous metabolic acidosis, nor was there evidence of encephalopathy or of proximal renal tubular dysfunction. Sodium excretion was significantly lower in the group of 9 patients than in either of the other two groups. Sodium sulfate and sodium phosphate infused after acid-loading rapidly reduced urine pH into the appropriately acidic range in the 9 patients. Incomplete distal renal tubular acidosis was thus delineated in 41% of the patients with alcoholic liver disease by means of a simple, safe, and short acid-loading test. Although the mechanism of this renal tubular acidifying defect remains unknown, a low distal delivery of sodium may have been partially responsible by limiting the transtubular potential difference.

6314 CELL-MEDIATED IMMUNITY TO LIVER IN PATIENTS WITH ALCOHOLIC HEPATITIS. *(E.)* Mihas, A. A.; Bull, D. M.; Davidson, C. S. (Boston City Hosp., Mass.). *Lancet* 1(7913):951-953, 1975.

The possibility that cell-mediated immunity to liver tissue is altered in alcoholic hepatitis was studied. Mononuclear cell responses to liver extracts were studied by a migration-inhibition assay in 24 patients with alcoholic liver disease (15 with acute alcoholic hepatitis and 9 with 'stable' Laennec's cirrhosis); 8 with viral hepatitis; 11 chronic alcoholics without evidence of liver disease; and in 15 healthy controls. Patients with acute alcoholic hepatitis demonstrated liver-antigen-induced inhibition of migration (migration index, 0.58), while patients with cirrhosis, alcoholism, and acute viral hepatitis, as well as controls, did not demonstrate such a response (migration indices, 0.92, 0.90, 0.86, and 0.99, resp.). It is concluded that cell-mediated immunity to normal or damaged liver tissue may act to perpetuate alcoholic hepatitis and thereby contribute to the development of cirrhosis.

6315 CHANGES IN THE RATE OF ALCOHOL CATABOLISM INDUCED BY PHENOBARBITAL, CHRONIC ALCOHOLISM, AND FASTING IN MAN. *(Ger.)* Bode, J. C.; Bode, C.; Thiele, D. (Med. Clin., Univ. Marburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:484-487, 1974.

The effect of long fasts (36 hr) and fructose on blood alcohol levels was studied in normal subjects whose average daily alcohol consumption was less than 20 g. Controls were given infusions of 0.5 g/kg alcohol over a 45-min period and, after a 30-min

equilibration period, blood samples were taken at 20 min intervals for 1 hr to determine alcohol concentrations. The rate at which blood alcohol levels decreased was 20-60% slower after a 36-hr than after a 12-hr fast. Infusions of fructose (0.8 g/kg/hr), given 2 hr after alcohol, stimulated decreases in blood alcohol levels of an average of 48% after a 12-hr fast and of 125% after a 36-hr fast. These findings suggest that decreased reoxidation of cytoplasmic NADH, rather than decreased alcohol dehydrogenase activity, is responsible for inhibiting alcohol oxidation. Administration of phenobarbital (300 mg/kg/day) for 6 days to these controls increased the rate of alcohol oxidation by an average of 51%, but values decreased significantly 2-3 days after discontinuation of the phenobarbital. This could be interpreted as indirect evidence for the existence of an alcohol-inducible microsomal ethanol-oxidizing system, but fructose-induced stimulation of alcohol oxidation was not significantly affected by administration of phenobarbital. This suggests that phenobarbital increases alcohol oxidation by a mechanism similar to that of fructose (increased NADH reoxidation). Studies performed on 13 alcoholic inpatients (average daily alcohol consumption of more than 100 g) showed that alcoholics metabolized alcohol more rapidly than controls (3.15 *versus* 2.51 mM/l). Values which were significantly above the normal range were found in only four of the 13 alcoholics who had abstained less than ten days. Fructose infusions caused little or no stimulation of alcohol oxidation in these four patients. This suggests that metabolic changes in the livers of chronic alcoholics cause an increase in the rate of alcohol metabolism *via* accelerated reoxidation of NADH.

6316 INHIBITORS OF LEUKOCYTE CHEMOTAXIS IN ALCOHOLIC LIVER DISEASE. *(E.)* Van Epps, D. E.; Strickland, R. G.; Williams, R. C., Jr. (Univ. New Mexico, Sch. Med., Albuquerque, N. M.). *Am. J. Med.* 59(2):200-207, 1975.

The characteristics of serum chemotactic inhibitory activity were determined and its relation to alcoholic liver disease and infection was examined in a study of 42 patients (37 men and 5 women, aged 23-68 yr) with alcoholic liver disease. Chemotaxis experiments were performed by the Boyden technique and chemotaxis was determined visually by counting the average number of cells migrating through the membrane per high power field for polymorphonuclear leukocytes or cells per oil immersion field for monocytes. The patients were also tested for lymphocytotoxic antibodies, relative proportions or absolute numbers of peripheral blood T and B lymphocytes, and serum immunoglobulin levels. Twenty-one patients showed serum chemotactic inhibitory activity. It was not related to any single biochemical or histologic feature in the patients studied. The frequency of chemotactic inhibitory activity was greatest in those with active infection. Serial studies demonstrated that chemotactic inhibitory activity may be a transient phenomenon, associated with active alcoholic liver disease or appearance of infection. Nine of 15 patients showed skin test anergy; chemotactic inhibitory activity was present in eight of these nine patients. Serum immunoglobulin A (IgA) and G (IgG) concentrations were significantly higher in patients with chemotactic inhibitory activity. Sucrose density gradient centrifugation of serums showing chemotactic inhibitory activity yielded three peaks of inhibitory activity: two had sedimentation coefficients of 10.7S and 6.8S, and the third was approximately 3S. The two higher molecular weight inhibitors were predominant in the 50% ammonium sulfate precipitate. Immunoabsorption by anti-IgA but not by anti-IgG or IgM columns removed the ammonium sulfate precipitable chemotactic inhibitors. The appearance of chemotactic inhibitors in patients with alcoholic liver disease may have relevance to their apparent susceptibility to serious infections.

6317 PLASMA LEVELS AND EXCRETION OF ESTROGENS IN URINE IN CHRONIC LIVER DISEASE. *(E.)* Pentikainen, P. J.; Pentikainen, L. A.; Azarnoff, D. L.; Dujovne, C. A. (Univ. Kansas Med. Cent., Kansas City, Kan.). *Gastroenterology* 69(1):20-27, 1975.

Plasma levels of nonconjugated esterone (E1), estradiol-17β (E2), and estriol (E3) and excretion of these estrogens in urine in conjugated and nonconjugated forms were measured by radioimmunoassay in 17 healthy males and 33 males with alcoholic liver disease. Total estrogens in urine were estimated after hydrolysis of the glucuronide and sulfate conjugates in hot acid. All samples were assayed in duplicate. Patients with liver disease had significantly elevated plasma levels of E1 (74.2 ± 8.0 SE *versus* 26.0 ± 1.7 pg/ml, $P < 0.001$), E2 (29.3 ± 2.2 *versus* 23.3 ± 2.0 pg/ml, $P = < 0.05$), and E3 (11.5 ± 1.9 *versus* 6.5 ± 0.7 pg/ml, $P < 0.01$). Excretion of total E1, E2, and E3 in urine was significantly increased in patients with liver disease. The increase was primarily in the nonconjugated fraction. Among those patients with chronic liver disease, the presence of ascites was associated with significantly higher plasma levels of E1 and excretion of larger amounts of E1 and E2 in urine. No differences were observed in the small number of patients with gynecomastia. The changes in plasma and urinary E1 and E2 values correlated directly with several liver function abnormalities, in particular with abnormal values for prothrombin time and plasma albumin levels. The increased plasma levels as well as the increased excretion of E1, E2, and E3 in urine observed in these patients are compatible with the explanation that they result from an increased rate of peripheral conversion of androgens.

6318 ALCOHOL AND DIETARY FACTORS IN CIRRHOSIS. AN EPIDEMIOLOGICAL STUDY OF 304 ALCOHOLIC PATIENTS. *(E.)* Patek, A. J., Jr.; Toth, I. G.; Saunders, M. G.; Castro, G. A. M.; Engel, J. J. (VA Hosp., Boston, Mass.). *Arch. Intern. Med.* 135(8): 1053-1057, 1975.

Alcohol intakes and dietary habits of 304 alcoholic, hospitalized patients were evaluated to determine whether differences exist in the drinking habits and dietary backgrounds of alcoholic patients with and without cirrhosis. Histories of alcohol intake were obtained using standard questionnaires. Dietary his-

tories over a 2-yr period were based on the recall method using a questionnaire for inquiry into ten food categories. There were 195 patients with hepatic cirrhosis, 40 precirrhotics and 69 noncirrhotics. In 85% to 94% of patients, excessive drinking began before the age of 30. Noncirrhotics were prone to drink less straight spirits than the other two groups combined, but the differences were not significant. More precirrhotics drank only spirits than did cirrhotics. Mean total daily calories ranged from 3200 to 3500, alcohol contributing 51% to 58%. The noncirrhotic group had a 13% higher intake of protein than the cirrhotic group and a 13% higher intake from other food sources. The precirrhotic groups was small, and the standard errors of its mean intakes were large so that it did not differ significantly from the other groups on statistical analysis. A familial incidence of alcoholism occurred in 60% of cirrhotics and in 58% of noncirrhotic alcoholics. The findings suggest that dietary factors may be involved in the pathogenesis of the disease.

6319 ZIEVE'S SYNDROME WITH CUTANEOUS MANIFESTATIONS. *(Fr.)* Heid, E.; Samsoeu, M.; Reuter, G.; Grosshans, E.; Busset, A. (Munic. Hosp. Strasbourg, France). *Ann. Dermatol. Syphiligr. (Paris)* 81(5):486-488, 1974.

6320 THE DIAGNOSTIC AND PROGNOSTIC VALUE OF THE IgA:TRANSFERRIN RATIO IN ALCOHOLIC CIRRHOSIS. *(Fr.)* Paris, J.; Jaillard, J.; Paris, J. C.; Sezille, G.; Fruchart, J. C.; Voiment, Y. M. (no affil.). *Lille Med.* 19(10):1002-1006, 1974.

6321 EVALUATIVE COMMENT ON THE RISK AND SAFETY OF AN ALIMENTARY SUBSTANCE: ALCOHOL. *(Fr.)* Tremolieres, J. (Lab. Biol. Natl. Conserv. Arts Sci., Paris, France). *Ann. Nutr. Aliment.* 28(4):317-334, 1974.

6322 SURGICAL CONSEQUENCES OF ALCOHOLISM. *(E.)* Orloff, M. J. (Dep. Surg., Univ. Calif., La Jolla). *Ann. N.Y. Acad. Sci.* 252:159-169, 1975.

6323 CURRENT POLICIES IN HEPATIC TRANSPLANTATION: CANDIDACY OF PATIENTS WITH ALCOHOLIC LIVER DISEASE OR PREFORMED ANTIDONOR ANTIBODIES AND A REAPPRAISAL OF BILIARY DUCT RECONSTRUCTION. *(E.)* Starzl, T. E.; Putnam, C. W.; Ishikawa, M.; Picacho, R.; Husberg, B.; Halgrimson, C. G.; Schroter, G. (Univ. Colorado Med. Cent., Denver). *Ann. N.Y. Acad. Sci.* 252:145-158, 1975.

6324 THE SKIN IN DIAGNOSIS OF ALCOHOLISM. *(E.)* Woeber, K. (Med. Fac., Univ. Bonn, West Germany). *Ann. N.Y. Acad. Sci.* 252:292-295, 1975.

6325 NEWER APPROACHES TO TREATMENT OF LIVER DISEASE IN THE ALCOHOLIC. *(E.)* Leevy, C. M.; Zetterman, R.; Smith, F. (New Jersey Med. Sch., Newark). *Ann. N.Y. Acad. Sci.* 252:135-144, 1975.

6326 CIRRHOSIS IN THE ALCOHOLIC AND ITS RELATION TO THE VOLUME OF ALCOHOL ABUSE. *(E.)* Lelbach, W. K. (Dep. Int. Med., Univ. Clin. Bonn, West Germany). *Ann. N.Y. Acad. Sci.* 252:85-105, 1975.

6327 LIVER DISEASE AND ALCOHOL: FATTY LIVER, ALCOHOLIC HEPATITIS, CIRRHOSIS, AND THEIR INTERRELATIONSHIPS. *(E.)* Lieber, C. S. (VA Hosp., Bronx, New York). *Ann. N.Y. Acad. Sci.* 252:63-84, 1975.

6328 ALCOHOLIC HEPATITIS, CIRRHOSIS, AND IMMUNOLOGIC REACTIVITY. *(E.)* Leevy, C. M.; Chen, T.; Zetterman, R. (New Jersey Med. Sch., Newark). *Ann. N.Y. Acad. Sci.* 252:106-115, 1975.

6329 THE ROLE OF ALCOHOLISM IN HEPATIC IRON STORAGE DISEASE. *(E.)* Powell, L. W. (Dep. Med., Univ. Queensland, Australia). *Ann. N.Y. Acad. Sci.* 252:124-134, 1975.

6330 THE PROTEIN BINDING OF SOME DRUGS IN PLASMA FROM PATIENTS WITH ALCOHOLIC LIVER DISEASE. *(E.)* Affrime, M.; Reidenberg, M. M. (Temple Univ. Sch. Med., Philadelphia, Pa.). *Eur. J. Clin. Pharmacol.* 8(3/4):267-269, 1975.

6331 THE SPECTRUM OF LIVER DISEASE IN ALCOHOLISM. *(E.)* Bhathal, P. S.; Wilkinson, P.; Clifton, S.; Rankin, J. G.; Santamaria, J. N. (St. Vincent's Hosp., Melbourne, Australia). *Aust. N.Z. J. Med.* 5(1):49-57, 1975.

6332 IMPAIRED DEFENSE MECHANISMS ASSOCIATED WITH ACUTE ALCOHOLISM. *(E.)* Johnson, Jr., W. D. (Cornell Univ. Med. Coll., New York, N.Y.). *Ann. N.Y. Acad. Sci.* 252:343-347, 1975.

See also, 5423, 6052, 6097, 6366.

6333 CHILDHOOD CIRRHOSIS ASSOCIATED WITH ALPHA-1-ANTITRYPSIN DEFICIENCY. A GENETIC, BIOCHEMICAL, AND MORPHOLOGIC STUDY. *(E.)* Asarian, J.; Archibald, R. W. R.; Lieberman, J. (Stanford Med. Cent., Calif.). *J. Pediatr.* 86(6):844-850, 1975.

Clinical and pathological data on a fatal case of juvenile cirrhosis due to homozygous α_1-antitrypsin deficiency followed over a 6-yr period, and comparisons of the phenotypic and biochemical findings in 27 relatives of the three-generation pedigree, are presented. At age 2 yr, 4 months, the proband (a girl) was hospitalized for evaluation of ascites. The liver and spleen were enlarged 3 cm below the costal margin. Serum transaminase, alkaline phosphatase, and LDH values were moderately elevated. Cholesterol concentration and serum ceruloplasmin were increased. At exploratory laparotomy the diffusely enlarged, firm nodular liver was biopsied: advanced cirrhosis of undetermined etiology was shown. Hepatosplenomegaly increased and a mild hyperbilirubinemia developed over the next 2 yr. By age 5 yr, examination showed spider angiomata, ascites, and a prominent venous pattern, with hepatosplenomegaly. At age 7 yr, 6 months she developed steatorrhea and massive ascites. Serum α_1-antitrypsin was less than 50 mg/dl, and a diagnosis of cirrhosis due to severe α_1-antitrypsin deficiency was made. She died at 8 yr, 5 months, of hepatic encephalopathy. A study of 27 relatives of the proband revealed 15 with heterozygous MZ phenotypes. Levels of circulating α_1-antitrypsin and trypsin-inhibiting capacity were shown to be unreliable in identifying the heterozygous state, protease inhibitor typing being necessary for definitive diagnosis. The morphologic evolution of the hepatic changes in this condition were studied, and the importance of the periodic acid-Schiff stain in identification of the characteristic cytoplasmic bodies is stressed.

6334 SODIUM SULFOBROMOPHTHALEIN KINETICS IN CIRRHOSIS OF THE LIVER BEFORE AND AFTER PORTACAVAL SHUNT. *(E.)* Cartel, G.; Benedetti, G.; Spine, G. P. (Dep. Surg. Pathol., Univ. Padua, Italy). *Am. J. Surg.* 130(7):47-50, 1975.

BSP kinetics in 36 patients (aged 17-65 yr) with liver cirrhosis and portal hypertension were investigated before and after portacaval shunt by the method of Barber-Riley *et al.* Side-to-side portacaval antastomosis was performed in 18 cases, end-to-side in 2, and splenorenal in the remaining 2 of the 22 patients who underwent operation. After i.v. injection of 5 mg/kg BSP, blood samples were taken from the opposite arm every 3 min for 20 min and thereafter every 5 min during the 30-120 min postinjection. In the 36 patients the percentage retention of BSP at 45 min was 29.95 ± 2.08; there was no overlap with the values for 39 healthy controls, in whom the upper limit of percentage retention was 3%. Preoperative BSP transfer rates, calculated from the plasma disappearance curves, differed significantly from control rates. Liver uptake of BSP and its transfer from liver to bile were grossly reduced and its transfer from liver back to plasma was augmented. In 15 patients 1 month after shunt, the liver uptake of BSP was 29.6% lower than preoperative values, whereas the transfer rate of BSP from liver to bile was unchanged. In 7 patients 1 yr after operation, the liver uptake of BSP was 28.3% lower and the transfer of BSP from liver to bile 29.9% lower than preoperative values. Further significant increases in BSP retention percentage occurred at 1 month and 1 yr. These results show that the increased percentage retention of BSP in patients with liver cirrhosis is accompanied by decreases in liver uptake of BSP and in transfer of BSP from liver to bile; transfer of BSP from liver to plasma is increased. One yr after operation, the deterioration of liver uptake of BSP ceases, while transfer of dye from liver to bile slightly deteriorates. Whether this deterioration is due to the operation or to a spontaneous evolution of cirrhosis could not be determined.

6335 EFFECT OF ORNITHINE ALPHA OXOGLUTARATE ON BRAIN METABOLISM IN PATIENTS WITH CHRONIC LIVER DISEASE. *(E.)* James, I. M.; Hamlyn, A. N.; Brant, P. C.; Hildrew, P. (R. Free Hosp., London, England). *J. Neurol. Neurosurg. Psychiatry* 38(3): 214-218, 1975.

The effect of ornithine α-oxoglutarate infusion on brain metabolism in 6 patients (4 men and 2 women, aged 24-70 yr) with chronic liver disease was studied with particular attention to glucose utilization. The patients had portal vein thrombosis (1); alcoholic cirrhosis (2); cryptogenic cirrhosis (2); and primary biliary cirrhosis (1). All exhibited only minimal neurological signs. Brain oxygen and glucose consumption were studied before and after i.v. infusion of ornithine α-oxoglutarate over a 15-min period. A further patient admitted in deep coma was also studied before and after 4-hr infusion with 60 g ornithine α-glutarate. Initial cerebral blood flow values ranged from 38-79 ml/100 g/min. There was no overall change in cerebral blood flow after infusion, but individually some changes were fairly substantial. Changes in $PaCO_2$ did not appear to have been the reason for these flow changes. Cerebral oxygen consumption increased significantly in 4 of the 6 patients, while cerebral glucose consumption decreased significantly in 5. After infusion in the patient with coma, cerebral blood flow and oxygen consumption increased and glucose consumption fell. These data confirm the finding of anaerobic glycolysis in patients with hepatic encephalopathy. In these patients, the biochemical disturbance of anaerobic glycolysis can be at least partially reversed with α-oxoglutarate administration, but the mechanism by which these changes are effected remains in doubt.

6336 COMPLICATIONS IN THE TREATMENT OF BLEEDING FROM ESOPHAGEAL VARICES WITH BALLOON TUBES. *(Ger.)* Brunswig, D.; Spech, H. J.; Heine, W. D. (Med. Clin., Univ. Wurzburg, Germany). *Therapiewoche* 24(39):4261-4262, 4264, 4267, 1974.

In comparison with the mortality (57%) resulting from bleeding esophageal varices in cirrhotics, severe, unavoidable complications associated with balloon tamponade are negligible. The most common complications are esophageal rupture, penetrating pressure ulcers, and aspiration during intubation. Fatal complications appear to be less frequent and bleeding is more often controlled when the Linton-Nachlas, rather than the Sengstaken-Blakemore, tube is used. Prospective studies are needed to determine whether selective, intra-arterial infusions of vasopressin are superior to balloon tamponade, particularly when extensive varices of the fundus are present. Although many complications can be avoided by the use of proper technique, trained personnel and intensive care are required to recognize and prevent untoward effects of tamponade. While tamponade can be a life-saving emergency measure in the treatment of bleeding esophageal varices, patients should be carefully evaluated for portacaval shunt surgery.

6337 PERIOD-AMPLITUDE ANALYSIS OF THE ELECTROENCEPHALOGRAM CORRELATED WITH LIVER FUNCTION IN PATIENTS WITH CIRRHOSIS OF THE LIVER. *(E.)* Kardel, T.; Stigsby, B. (Gentofte Hosp., Univ. Copenhagen, Denmark). *Electroencephalograph. Clin. Neurophysiol.* 38(6):605-609, 1975.

Studies of the activity distribution of the electroencephalogram (EEG) by period-amplitude analysis were made in 40 patients with liver cirrhosis, with or without a surgical portacaval anastomosis, and in 25 controls. From the two parieto-occipital leads, 100 sec of EEG were examined. A quantitative determination of liver function was made in the patients by the galactose elimination capacity. A gradual slowing of the EEG was observed when passing from controls to nonshunted cirrhotics and shunted cirrhotics. This was reflected by significant differences in the amount of activity in some of the frequency bands and by a decrease of the dominant EEG frequency. There was a statistically significant, although weak, positive correlation between the dominant EEG frequency and the galactose elimination capacity in the non-shunted patients and in both patient groups considered together; the correlation was not significant in the shunted patients considered alone. A similar shift to lower EEG frequencies was previously observed in patients with chronic renal failure. Thus, it is concluded that the slowing of the EEG in patients with cirrhosis of the liver is an unspecific expression of metabolic brain disturbance.

6338 CHANGES IN PLASMA VISCOSITY IN CHRONIC LIVER DISEASE. *(Ger.)* Palme, G.; Kochen, M.; Leonhardt, H. (Steglitz Clin., Free Univ., Berlin, Germany). *Clin. Chim. Acta* 61(2):163-169, 1975.

Measurements of plasma viscosity were performed on fasting blood samples from 37 patients (20 men and 17 women, mean age 56 yr) with chronic hepatitis (six cases), chronic hepatitis evolving into cirrhosis (seven cases), ective cirrhosis (six cases), and inactive cirrhosis (17 cases). Portal hypertension was diagnosed in 25 cases and esophageal varices in seven. On the basis of these findings, patients were classified into three groups. Decreased viscosity was found in patients with cirrhosis, severe portal hypertension, and esophageal varices. Viscosity was normal in patients with inactive cirrhosis without portal hypertension. Patients with active cirrhosis and chronic progressive hepatitis had increased viscosity. Increased plasma viscosity was associated with increased concentrations of IgG, while decreased viscosity was associated with low fibrinogen levels. Close correlations were also found between plasma viscosity and the activities of SGOT, SGPT, and GLDH. When 5 patients with chronic progressive hepatitis and increased IgG concentrations were treated with corticosteroids, the plasma and whole-blood viscosities decreased to normal values with a parallel decrease in SGOT activity as a manifestation of restored hepatocellular integrity and an improvement in the normotest (factors II, VII and X) as a manifestation of improved synthesis of liver-specific clotting factors. Further research is needed to determine whether blood viscosity values can be used as an indication for corticosteroid therapy in chronic liver disease.

6339 ALCOHOL AND LIVER CIRRHOSIS-A QUANTITATIVE PROBLEM? *(Ger.)* Lelbach, W. K. (Med. Clin., Univ. Bonn, Germany). *Therapiewoche* 24(51): 6025-6032, 1974.

The author's experience with a group of 526 male alcoholics, treated for 6 months as inpatients, showed that it was easier to ascertain the duration of alcohol abuse than to establish the daily consumption. The mean duration of alcohol abuse was 9.1 yr, but only 417 patients gave reliable information on their daily consumption; 186 (45%) reported amounts of 160 g or less, while 231 (55%) reported amounts above 160 g. The mean for the entire group was 180 g/day. On the basis of biopsy findings in 320 cases and clear-cut clinical diagnoses of cirrhosis in 14, patients were divided into four groups according to the duration of alcohol abuse. Since the daily consumption increased with increasing duration of abuse, the effect of these two factors could not be evaluated separately. Consequently, 216 patients with identical durations of abuse (about 8 yr) were subdivided into two groups with different daily consumptions. The age distribution and body weights were identical in these two groups. In patients consuming more than 160 g/day (mean 227 g), only 10% had completely normal histological findings, and precirrhotic changes were twice as common (33% *versus* 14%) as in patients consuming less than 160 g/day. While none of the patients in the second group had cirrhosis, cirrhosis was diagnosed in 14% of those consuming more than 160 g/day. Thus, the risk of developing alcoholic cirrhosis depends upon both the duration of alcohol abuse and the amount of alcohol consumed/day. In a group of 265 biopsied alcoholics, the average daily consumption was related to body weight and time in hours, and this was plotted against the extent of histological

change. The resulting curve was almost a straight line. In this group of 265 biopsied alcoholics, the product of dose/kg body weight and the duration of abuse was calculated for each patient and ranked into 13 classes, and the incidence of cirrhosis and precirrhotic changes was determined for each class. Regression analysis demonstrated a highly significant correlation between the total dose of alcohol consumed/kg and the incidence of both cirrhosis and liver damage more severe than uncomplicated fatty liver.

6340 INSORPTION OF TRITIATED WATER IN CIRRHOSIS. *(Ger.)* Mertz, D. P.; Weiss, M. (Med. Polyclin., Univ. Freiburg, Germany). *Res. Exp. Med. (Berl.)* 165(1):9-18, 1975.

The insorption of tritiated water and its distribution volumes were studied in ten patients with cirrhosis (seven men and three women, aged 37-66 yr) and five normal controls after p.o. administration of 200 μCi of THO in an average of 100 ml water. Cirrhosis was alcoholic in etiology in four cases and was associated with mild diabetes mellitus in three cases and with hemochromatosis in one. Both blood and urine samples were taken before and blood samples were taken at 10-min intervals until 2 hr after the administration of THO. Then blood and urine samples were taken at hourly intervals between 3 and 24 hr after administration of THO. The time required for THO to reach its maximum concentration in the blood varied greatly (from 30-110 min) in cirrhotics and was not related to the volume of total body water or to the etiology of the cirrhosis. Although the time required to reach maximum THO concentrations was normal in three cirrhotics, it was significantly longer (mean 69.0 min) in cirrhotics than in controls (37.0 min). The volume of total body water related to weight was significantly greater (59.8%) in cirrhotics than in controls (52.8%). For the first 20 min after administration of THO, the rate at which THO was insorbed was the same in cirrhotics and controls. Values for this rate were significantly greater in cirrhotics after 40, 50, and 70 min. Although changes in fluid balance cannot be ruled out as a cause of slower insorption of THO in cirrhotics, it is suggested that changes in intestinal blood flow, tissue pressure, and colloid-osmotic pressure contribute to this abnormality.

6341 THE SYSTEMIC CIRCULATION IN PORTAL HYPERTENSION. *(Ger.)* Liehr, H.; Grun, M.; Thiel, H. (Med. Clin., Univ. Wurzburg, Germany). *Z. Gastroenterol.* 13:133-146, 1975.

Experimental and clinical studies were performed to determine whether the development of hyperdynamic circulation and disorders in volume regulation is a process associated with portacaval collaterals or with cirrhosis, and whether circulatory changes occurring in cirrhosis represent a mechanism compensating for changes in portal blood flow. In rats in which portal hypertension was gradually induced by occlusion of the portal vein with Ameroid rings, the cardiac output increased to a mean of 36.9 ml/min/100 g (control value was 6.3 ml/min), and the circulation time decreased to 2.8 sec (control value was 3.6 sec) with increasing shunt volume (64%). The blood volume was 7.5 ml/100 g in comparison with 6.3 ml/100 g for controls. These changes regressed with a decrease in the portacaval shunt volume caused by the formation of portahepatic anastomoses. Comparable values were obtained in rats with portacaval anastomoses 28 days after surgery. In 77 patients with cirrhosis, the cardiac output was 3.3 l/min/m^2 for those with few collaterals and 4.1 l/min/m^2 for those with pronounced collaterals. Similarly, a significant difference was found between the arm-ear time in patients with few collaterals (12.6 sec) and those with pronounced collaterals (10.2 sec). The blood volume increased significantly (from 74.2 ml/kg to 36.0 ml/kg) as the extent of portacaval collaterals increased. The ratio of blood flow to liver mass remained normal (shunt volume 64%), or decreased to 75% of the normal values (shunt volume 100%), as a result of increased cardiac output. It is concluded that hyperdynamic circulation and hypervolemia are only associated with a hemodynamically relevant portacaval collateral circulation and are independent of cirrhosis. These conditions develop independent of the portal pressure and can be considered to compensate for decreases in portal blood flow.

6342 VALUES OF LARGE DOSES OF PREDNISOLONE IN THE TREATMENT OF HEPATIC COMA IN CIRRHOTICS FOLLOWING SHUNT SURGERY. *(Ger.)* Paquet, K. J.; Figge, H.; Welte, D. (Surg. Clin., Univ. Bonn, Germany). *Therapiewoche* 24(42):4817-4820, 4823-4826, 1974.

Large doses of prednisolone were administered over a short period to 26 patients who developed any of the following indications after shunt surgery: (1) liver coma, no matter what the cause, (2) acute exacerbation of cirrhosis, or (3) increased fibrinolysis or disseminated intravascular clotting. In addition, prednisolone was given to patients with small livers and adequate functional reserves who required end-to-side portacaval shunts and to patients with decompensated liver function who underwent emergency surgery. Of these 26 patients, 16 had portacaval shunts and 10, distal splenorenal shunts; six underwent emergency surgery. Prednisolone was administered in doses of 100 mg/24 hr on days 1-4 postoperatively; 50 mg/24 hr on days 5-6; 100-300 mg/24 hr on days 7-8; and 10-100 mg/24 hr on days 9-14. Controls consisted of 50 patients who underwent shunt surgery and who, for the most part, developed no postoperative complications. Of the 26 prednisolone-treated patients, five died: the cause of death was liver coma with recurrent bleeding (2 cases), liver coma without recurrent bleeding (1), subphrenic abscess, local peritonitis, and septicopyemia following splenorenal shunt surgery (1), and pneumonia and heart failure (1). Among patients given large doses of prednisolone, SGOT and SGPT values which were significantly increased immediately after surgery normalized within 14-21 days; similar data were not available from the control group. The increase in bilirubin levels which occurred in the first two days after surgery was

significantly higher among prednisolone-treated patients than among controls. Normalization of total protein and cholinesterase levels and clotting factors occurred within 10-14 days in prednisolone-treated patients. None of the prednisolone-treated patients developed side effects. Although these findings suggest that large doses of prednisolone may prevent liver coma from developing after shunt surgery, no definitive conclusions can be drawn because of the small number of patients treated and because of the many other therapeutic measures utilized to prevent liver failure (e.g., bed rest, high-carbohydrate diet, gastric suction, maintenance of fluid and electrolyte and acid-base balance, administration of antibiotics, albumin, plasma substitutes, aldosterone antagonists, lactulose).

6343 INCREASED FIBRINOGEN SYNTHESIS IN PORTAL HYPERTENSION. *(Ger.)* Grun, M.; Liehr, H.; Thiel, H.; Brunswig, D.; Buchenau, D. (Med. Clin., Univ. Wurzburg, Germany). *Z. Gastroenterol.* 12(suppl.):409-410, 1974.

Portal hypertension, portacaval anastomoses, hypoxic atrophy of the liver, and hypervolemia with hypercirculatory syndrome were induced in rats by placing rings of expandable material around the portal vein. Six days after surgery the plasma fibrinogen level was still normal, but the mean blood volume had increased to 148% of control values, the plasma fibrogen pool to 154% of control values, and the rate of incorporation of ^{14}C-leucine into thrombin-precipitated plasma protein/g liver to 211% of control values. During normalization of the plasma volume by formation of portohepatic anastomoses (21 days after surgery), the rate at which ^{14}C-leucine was incorporated into thrombin-precipitated plasma protein decreased to 61% of the control values. Similar studies were performed on 28 patients with cirrhosis who were divided into three groups, depending upon the extent to which portacaval anastomoses had developed. In the group as a whole, plasma volume was increased. There was a positive correlation between this increase and the extent of the portacaval shunt volume. The hepatic volume decreased with increasing portacaval shunt volumes. Since the mean plasma fibrinogen level for the entire group was 223 ± 72 mg/100 ml, the plasma fibrinogen pool was increased to 8.23 ± 2.49 g. However, the extent of this increase differed in each of the three groups and was not related to hepatic volume. Thus, in both experimental animals and patients with cirrhosis the plasma fibrinogen pool is increased despite a decrease in the hepatic volume, suggesting that the rate of fibrinogen synthesis is increased. Therefore, the plasma fibrinogen pool is apparently regulated by the plasma fibrinogen level, even under pathological conditions.

6344 PIGMENTED CORNEAL RINGS IN A PATIENT WITH PRIMARY BILIARY CIRRHOSIS. *(E.)* Fleming, C. R.; Dickson, E. R.; Hollenhorst, R. W.; Goldstein, N. P.; McCall, J. T.; Baggenstross, A. H. (Mayo Clin. Mayo Found., Rochester, Minn.). *Gastroenterology* 69(1):220-225, 1975.

A case report of a 54-yr-old woman with primary biliary cirrhosis in whom corneal rings similar to Kayser-Fleischer rings were detected by slit-lamp biomicroscopy is presented. Jaundiced serum was first noted in 1966. In 1969 a percutaneous needle biopsy of the liver showed marked inflammation of portal tracts with severe piecemeal necrosis, marked periportal and septal fibrosis, and a decrease in the number of interlobular bile ducts. The patient was treated with cholestyramine and fat-soluble vitamins. In September 1973 excretory urography confirmed a radioopaque renal stone, and the urine oxalate excretion was 60 mg/24 hr (normal <25 mg/24 hr). In May 1974 the patient showed high levels of serum ceruloplasmin, 97 mg/dl and repeated 88 mg/dl (normal 22.9-43.1); serum copper, 279 µg/dl (normal 75-145); urinary copper excretion, 221 µg (normal 15-49); and hepatic copper concentrations, 756 µg/g of dry weight of liver (normal 15-35). Needle biopsy of the liver revealed the cirrhotic stage of primary biliary cirrhosis with reduction in interlobular bile ducts and moderate atypical ductular proliferation. Results of ophthalmological examination were normal except for severe scleral icterus and 1.5-mm wide band of yellowish green pigmentation that extended around the entire periphery of each cornea and in the region of Descemet's membrane. The ring was denser peripherally and faded to normal cornea away from the limbus. The color and location of the pigment were like those of Kayser-Fleischer rings, but were of lesser density than is usual in patients with advanced Wilson's disease. Kayser-Fleischer rings, similar to those seen in this patient with primary biliary cirrhosis, have been observed in patients with early or minimal manifestations of Wilson's disease. Wilson's disease was excluded on the basis of the elevated serum ceruloplasmin and copper levels, the lack of neurological disease despite advanced liver disease, a negative family history and the typical clinical features of primary biliary cirrhosis. The patient was treated with a low-sodium diet, diuretics, cholestyramine and vitamin K.

6345 EFFECT OF LIVER FAILURE ON THE RESPONSE OF VENTILATION AND CEREBRAL CIRCULATION TO CARBON DIOXIDE IN MAN AND IN THE GOAT. *(E.)* Stanley, N. N.; Salisbury, B. G.; McHenry, L. C., Jr.; Cherniack, N. S. (Univ. Pennsylvania Sch. Med., Philadelphia, Pa.). *Clin. Sci. Mol. Med.* 49(2): 157-169, 1975.

The acid base state of arterial blood and cerebrospinal fluid and the ventilatory response to inhaled CO_2 were evaluated in 12 patients with liver disease and the effect of liver failure in unanesthetized goat was determined. The pH, oxygen partial pressure (P_{O2}) and CO_2 partial pressure (P_{CO2}) in blood and cerebrospinal fluid of the patients were measured with appropriate electrodes, and arterial and cerebrospinal fluid bicarbonate concentrations were calculated from the measured P_{CO2} and pH values. The ventilatory response to CO_2 was evaluated in both patients and animals by a modification of the rebreathing method. Liver failure was accompanied by a respiratory alkalosis in both the patients and in the goats. The P_{CO2} was reduced and pH increased

in the arterial blood; plasma bicarbonate concentrations were reduced in keeping with a partially compensated respiratory alkalosis. In the cerebrospinal fluid there was also a reduction in P_{CO_2}, a slight increase in pH, and a reduction in bicarbonate concentration. The slope of the ventilatory response to CO_2 was reduced when liver failure was severe, in patients and goats alike. There was a reduction in the extrapolated P_{CO_2} at zero ventilation, even when liver failure was mild. The mean cerebral blood flow fell by 30% after the onset of liver failure as compared with that in the control period, whereas cerebral vascular resistance increased by 40%. These changes were associated with hypocapnia, the mean arterial P_{CO_2} falling by nearly 10 mm Hg. The cerebral metabolic rate for O_2 fell by 22%. The dangers of hypercapnia in hepatic cirrhosis are confirmed by the fall in cerebral metabolic rate for O_2 induced by CO_2 breathing in the goats with liver failure. The possible clinical relevance of this adverse response to exogenous CO_2 is that cerebral function may be similarly impaired when a patient with liver failure is exposed to respiratory depressants.

6346 RESPONSE TO DOPAMINE HYDROCHLORIDE IN THE HEPATORENAL SYNDROME. *(E.)* Bennett, W. M.; Keeffe, E.; Melnyk, C.; Mahler, D.; Rosch, J.; Porter, G. A. (Univ. Oregon Med. Sch., Portland). *Arch. Intern. Med.* 135(7):964-971, 1975.

Seven patients with hepatorenal syndrome were studied before and after dopamine hydrochloride treatment (initially 2 µg/kg/min, increasing to 4-6 µg/kg/min intra-arterially and subsequently i.v.) to determine the effects of dopamine on the renal cortical vasculature and cortical blood flow rate. Five patients with decompensated cirrhosis of the liver without renal failure were also studied but were not given dopamine. Renal blood flow was measured by analysis of the washout curve of xenon 133 and selective arteriography was used to determine the status of the renal cortical vasculature. Renal pathological material was available from three cases at necropsy. Mean renal blood flow and flow rate through the cortical component were decreased in both groups of cirrhotic patients compared with a control group of normal transplant donors. Both cirrhotic groups also showed redistribution of blood from the cortex to juxtamedullary areas. After intra-arterial dopamine treatment in the hepatorenal patients, the cortical blood flow rate increased significantly, with insignificant increases in the mean renal blood flow and percent of total blood flow in component I. The antiographic findings paralleled the xenon Xe 133 curves in the hepatorenal patients and showed diffuse cortical vasospasm; in the five patients with no renal failure, the angiographic findings were within normal limits. Dopamine treatment improved the angiographic appearance of the renal cortical vasculature in the hepatorenal group. However, little improvement in renal function was noted after the i.v. dopamine infusions; urine osmolarity increased significantly and sodium excretion decreased significantly after i.v. dopamine, but changes in the urine volume and glomerular filtration rate were insignificant. Two patients survived the hepatorenal syndrome and left the hospital with normal renal function as an apparent result of dopamine treatment. No adverse reactions were noted as a result of the dopamine infusions. Renal histopathologic findings were normal in the three necropsy specimens. It is concluded that standard treatment involving symptomatic treatment of the liver disease, exclusion of the renal causes of azotemia, and intravascular volume repletion should be used in the treatment of hepatorenal syndrome.

6347 MASSIVE SPONTANEOUS PORTAL-SYSTEMIC SHUNTING WITHOUT VARICES. *(E.)* Wexler, M. J.; MacLean, L. D. (Dep. Surg., R. Victoria Hosp., Montreal, Canada). *Arch. Surg.* 110(8):995-1033, 1975.

Six patients, some with previously unsuspected liver disease or portal hypertension, with massive spontaneous portal-systemic shunts were identified by umbilical vein portal phlebography. In two patients, the shunt was between the splenic and left renal vein, while in another it was between the coronary and left renal veins. In two patients, a spontaneously reopened and patent umbilical vein served as a massive conduit anastomosing with the iliac system. In one patient, the shunt was via the inferior mesenteric vein to the hemorrhoidal plexus. None of these patients had esophageal varices and all but one were free of gastrointestinal bleeding. The spectrum of clinical manifestations varied greatly. Two totally asymptomatic patients were being investigated for splenomegaly and a third for epigastric pain; none was known to have liver disease. One patient had severe encophalopathy, while another with well-established asymptomatic liver disease since childhood was admitted with upper gastrointestinal bleeding of obscure cause. Five patients clearly demonstrated hepatofugal portal flow, while the sixth showed noticeable reduction of the portal fraction of total hepatic blood flow. All patients had small, shrunken livers, enlarged spleens, and demonstrated a striking absence of the plethora of collateral veins usually seen on venus portography in patients with portal hypertension. The spontaneous shunts resembled surgically constructed portal-systemic shunts in magnitude, and perhaps in volume of flow. Liver biopsies showed Laennec's cirrhosis in two patients, postnecrotic cirrhosis in two, and chronic aggressive hepatitis with cirrhosis in two. Spontaneous shunts may be more common than suspected in cirrhotics and may explain the lack of variceal hemorrhage in many patients.

6348 PORTAL AND SYSTEMIC EFFECTS OF SELECTIVE INFUSION OF VASOPRESSIN INTO THE SUPERIOR MESENTERIC ARTERY IN CIRRHOTIC PATIENTS. *(E.)* Millette, B.; Huet, P. M.; Lavoie, P.; Viallet, A. (Hospital Saint-Luc, Montreal, Canada). *Gastroenterology* 69(1):6-12, 1975.

The portal and systemic effects of a selective infusion of vasopressin into the superior mesenteric artery were evaluated in eight male cirrhotic patients with portal hypertension, who underwent umbilicoportal catheterization. Vasopressin was infused selectively into the superior mesenteric artery at a rate

of 0.2 U/0.75 cc/min with a constant rate infusion pump for 40–60 min. Free portal venous pressure decreased significantly from 27.0 ± 1.4 mm Hg to 24.4 ± 1.4 mm Hg (9.6%). In all patients, portal oxygen pressure decreased significantly with a mean fall of 18.8%. Femoral arterial oxygen pressure decreased significantly (7.3%) while the mean femoral arterial blood pressure increased significantly (18.95) in all patients. Variable changes of the cardiac output were observed in the seven patients during the vasopressin infusion. In two patients, the cardiac output increased by 27 and 15%; in two patients, it was minimally decreased (1 and 8%), but in the other three patients it was markedly decreased by 35, 18 and 27%. It is concluded that selective infusion of vasopressin into the mesenteric circulation does not induce a marked decrease of the portal venous pressure in cirrhotic patients with portal hypertension undergoing umbilicoportal catheterization. In any controlled clinical trial, the risk of venous and/or arterial thrombosis, the decrease of portal oxygen pressure and possible systemic effects have to be considered in cirrhotic patients with ruptured varices.

6349 NEGATIVE INFLUENCES OF ASCITES ON THE CARDIAC FUNCTION OF CIRRHOTIC PATIENTS. *(E.)* Guazzi, M.; Polese, A.; Magrini, F.; Fiorentini, C.; Olivari, M. T. (Inst. Cardiovasc. Res., Milan, Italy). *Am. J. Med.* 59(2):165-170, 1975.

The negative influences of ascites on the cardiac function of 21 hospitalized men with Laennec's cirrhosis were investigated. Hemodynamic measurements were carried out with the patients in the fasting state, in the supine position and without premedication, at each of the following times: in the steady state and, resp., after removal of 500, 1000, 2000, 3000, 4000 and 5000 ml of ascitic fluid. The paracentesis was continued to remove as much fluid as could be obtained; in two patients it was 8000 ml. The hemodynamic pattern during paracentesis was characterized by no significant change in heart rate, systolic and diastolic arterial pressure, or pulmonary mean arterial and wedge pressures; progressive increase in cardiac output, stroke volume, stroke work, and mean rate of ejection of the right and left ventricle; increasing reduction in intra-abdominal pressure and in the inferior vena cava and right atrial pressures. Paracentesis was thus followed by circulatory variations which were primarily represented by improvement of right and left ventricular function, as clearly indicated by the significant increase in cardiac output exclusively due to an increment in stroke volume, by the rise in stroke work, and mean rate of systolic ejection of both sides of the heart. The improvement in cardiac function was associated with an increasing reduction in intra-abdominal, inferior vena cava and right atrial pressures. There was a direct relation between the decrease in systolic right atrial pressure and the increase in cardiac output at measured withdrawal of ascitic fluid. The average variations in systolic right atrial pressure were related to the average changes in right ventricular stroke work at measured drainage of ascites. The high caval and right atrial pressures along with the elevated abdominal pressure and the response to paracentesis suggest a mechanism in which tense ascites increase the hydrostatic intra-abdominal pressure acting on the diaphragm; this raises the intrapleural pressure, which, in turn, reduces the effective filling pressure of the heart and shifts the cardiac output curve to the right.

6350 STUDY OF PORTAL CIRCULATION WITH THE AID OF THE GAMMA CAMERA. *(Fr.)* Roucayrol, J. C.; Paraf, A.; Syrota, A.; Vinot, J. M. (Cochin Hosp., Paris, France). *Bordeaux Med.* 8(6):623-624, 1975.

6351 CIRRHOTIC ASCITES. *(Fr.)* Aron, E. (no affil.). *Ouest. Med.* 28(3):131-133, 1975.

6352 THE EFFECTS OF SOME HEPATIC FUNCTION INDICES ON THERAPY WITH UDPG, GLUTATHIONE AND VITAMIN B_{12} IN CIRRHOTICS. *(It.)* Coltorti, M. (Fac. Med. Chir. I, Univ. Naples, Italy). *Clin. Ter.* 72(4):323-328, 1975.

6353 NUTRITIONAL PROBLEMS IN CIRRHOSIS AND FOLLOWING PORTACAVAL ANASTOMOSES. *(Fr.)* Joubaud, F. (Univ. Hosp. Cent., Angers, France). *Anesth. Analg. (Paris)* 31(6):875-888, 1974.

6354 ALTERATION IN THE ACID-BASE BALANCE IN LIVER CIRRHOSIS. *(It.)* Berni, G.; Lagi, A. (Clin. Gen. Med., Med. Ther., Univ. Florence, Italy). *Recent. Prog. Med. (Roma)* 56(3):230-237, 1974.

6355 GAS CHROMATOGRAPHY URINARY STEROID PROFILE IN HEPATIC CIRRHOSIS. *(It.)* Molino, G.; Gaidano, G.; Berta, L.; Avagnina, P.; Marinone, C. (Inst. Semeiot. Med., Univ. Turin, Italy). *Boll. Soc. Ital. Biol. Sper.* 50(20):1662-1667, 1974.

6356 METASTATIC CARCINOMA IN CIRRHOTIC LIVER, STATISTICAL SURVEY OF AUTOPSIES IN JAPAN. *(E.)* Hamaya, K.; Hashimoto, H.; Maeda, Y. (Okayama Saiseikai Gen. Hosp., Japan). *Acta Pathol. Jpn.* 25(2):153-159, 1975.

6357 AUSTRALIA ANTIGEN AND MALIGNANT HEPATOMA. *(E.)* Theodoropoulos, G.; Archimandritis, A.; Angelopoulos, B. (Med. Sch., Univ. Athens, Greece). *Ann. Int. Med.* 82(6):809-810, 1975.

6358 UNILATERAL NEVOID TELANGIECTASIA WITH GASTRIC INVOLVEMENT. *(E.)* Anderton, R. L.; Smith, Jr., J. G. (Med. Coll. Georgia, Augusta). *Arch. Dermatol.* 111(6):617-621, 1975.

6359 FLUID RETENTION IN LIVER DISEASE. *(E.)* Blendis, L. M. (Central Middlesex Hosp., London, England). *Br. J. Hosp. Med.* 13(1):47-54, 1975.

6360 ARTERIAL PORTOGRAPHY IN THE MANAGEMENT OF PORTAL HYPERTENSION. *(E.)* Legge, D. A.; Whelton, M.; Hennessy, T. (St. Finbarr's Hosp., Cork, England). *Ir. J. Med. Sci. (Dublin)* 144(3):92-101, 1975.

6361 CIRRHOSIS OF THE LIVER. *(E.)* McIntyre, N. (R. Free Hosp., London, England). *Br. J. Hosp. Med.* 13(1):8-16, 1975.

6362 SPLENIC PORTOGRAPHY IN THE EVALUATION OF THE MESOCAVAL SHUNT WITH A DACRON PROSTHESIS. *(It.)* Peracchia, A.; Morosini, S.; Roncoroni, L. (Inst. Clin. Gen. Surg., Surg. Ther., Parma, Italy). *Ateneo Parmense [Acta Biomed.]* 45(3):93-111, 1974.

6363 PRESINUSOIDOL HEPATIC ARTERIOPORTAL SHUNT. ANGIOGRAPHIC VISUALIZATION IN LIVER CIRRHOSIS. *(Fr.)* Osteaux, M.; Jeanmart, L. (Radiodiagn. Serv., Bordes Inst., Brussels, Belgium). *J. Belg. Radiol.* 57(3):205-210, 1974.

6364 RECENT ASPECTS OF THE TREATMENT OF PORTAL HYPERTENSION IN CIRRHOTICS. *(Fr.)* Kestens, P. J.; Lambilliotte, J. P. (Clin., Univ. St. Pierre, Louvain, Belgium). *Acta Gastroenterol. Belg.* 37(9): 383-401, 1974.

6365 PREDICTING THE CAUSE OF GASTROINTESTINAL BLEEDING IN CIRRHOTICS AS A FUNCTION OF THE EXTENT OF HEPATOCELLULAR INSUFFICIENCY. *(Fr.)* Franco, D.; Deporte, A.; Bismuth, H. (Paul Brousse Hosp., Villejuif, France). *Biol. Gastroenterol. (Paris)* 7(4):330-331, 1974.

6366 THE FATE OF AMINO ACIDS PERFUSED IN MALNOURISHED ALCOHOLICS WITH CIRRHOSIS. *(Fr.)* Michel, H.; Raynaud, A.; Mourrut, B.; Bellet, H.; Sany, C.; Bertrand, L. (St. Eloi Hosp., Montpellier, France). *Biol. Gastroenterol. (Paris)* 7(4):329, 1974.

6367 TREATMENT OF LIVER COMA AND HEPATIC ENCEPHALOPATHY. *(Ger.)* Lelbach, W. K. (Med. Clin., Univ. Bonn, Germany). *Med. Welt* 25(46):1921-1926, 1974.

6368 AN UNUSUAL SCINTIGRAPH OF LIVER CIRRHOSIS. *(Ger.)* Dressler, J.; Kramann, B. (Clin. Polyclin. Nucl. Med., Tech. Univ. Munich, Germany). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 122(2):174-175, 1975.

6369 THE BUDD-CHIARI SYNDROME: SYMPTOMATOLOGY IN A YOUNG WOMAN WITH A POSTHEPATIC BLOCK. *(Ger.)* Deeg, P.; Schneider, K. W.; Jesse, R. (Med. Clin., Univ. Wurzberg, Germany). *Med. Monatsschr.* 29(2):78-82, 1975.

6370 ENCEPHALOPATHY WITH ACQUIRED CHRONIC CIRRHOSIS OF THE LIVER. *(Ger.)* Noetzel, H.; Weber, M. (Inst. Pathol., Univ. Freiburg, Germany). *Med. Welt* 26(4):163-166, 1975.

6371 CLINICAL EVALUATION OF THE SERUM IRON CONCENTRATION IN CHILDHOOD CIRRHOSIS. *(Rus.)* Plaksina, G. B.; Dedikova, L. A. (M. F. Vladimirskii Reg. Res. Inst. Clin. Med., Moscow, USSR). *Pediatriia* (3):61-62, 1975.

6372 SURGICAL SURVIVAL IN PATIENTS WITH CIRRHOSIS ASSOCIATED WITH PORTAL HYPERTENSION. *(Rus.)* Dushanin, S. A.; Ekzarkhov, B.A.; Morev, A. G.; Boichuk, G. K.; Molchanov, A. I. (Kharkov Res. Inst. Gen. Emergency Surg., USSR). *Klin. Khir.* (2):45-49, 1975.

6373 THERAPY OF DECOMPENSATED CIRRHOSIS. *(It.)* Albano, O.; Francavilla, A.; Leo, M.; Centonze, M.; Salerno, M. T. (no affil.). *Rass. Clin. Sci.* 50(1):1-10, 1974.

6374 THERAPY OF COMPENSATED CIRRHOSIS. *(It.)* De Pergola, E. (no affil.). *Rass. Clin. Sci.* 50(1):11-15, 1974.

6375 ETIOLOGICAL DIAGNOSIS OF GASTROINTESTINAL HEMORRHAGE IN CIRRHOTICS. *(Por.)* Miniconi, P. (no affil.). *J. Med. (Lisboa)* 87(1642): 174-176, 1975.

See also, 5415, 5694, 6086, 6094, 6097, 6100, 6105, 6127, 6187, 6236, 6245, 6246, 6273, 6277, 6289, 6292, 6296, 6297, 6300, 6301, 6328.

LIVER AND BILIARY TRACT

Gallbladder and Biliary Tract Diseases

6376 PROLONGED SECRETION OF LITHOGENIC BILE AFTER CHOLECYSTECTOMY. *(E.)* McDougall, R. M.; Walker, K.; Thurston, O. G. (Dep. Surg., Univ. Alberta, Edmonton, Canada). *Ann. Surg.* 182(2):150-153, 1975.

The possible secretion of a lithogenic bile following cholecystectomy for cholesterol gallstones was studied in eight patients 1.5 to 23 yr after cholecystectomy for presumed cholesterol gallsones. The cholesterol, lecithin, and bile acid levels in the hepatic

bile of these subjects were compared with the levels in the gallbladder bile of 15 patients currently undergoing cholecystectomy for pure and mixed cholesterol gallstones in functional gallbladders. Bile was obtained by needle or T-tube aspiration of the common bile duct, duodenal catheter aspiration, or *via* gastrostomy tube. The hepatic bile samples for the subjects who had undergone cholecystectomy 1.5 to 23 yr previously were saturated or supersaturated with cholesterol in all cases, the mean lithogenic index for this group being 1.31. The bile in the 15 patients currently undergoing cholecystectomy was supersaturated with cholesterol in all but three cases, the mean lithogenic index being 1.41. The mean value of molar percent cholesterol in the current cholecystectomy group was 14.0, while that in the previous cholecystectomy group was 12.7. In the previous cholecystectomy group, bile supersaturated with cholesterol did not become less saturated with time. The data support the concept that, once begun, the production of a lithogenic bile by the liver is a lifelong phenomenon.

6377 STUDY OF CHOLELITHIASIS ASSOCIATED WITH CHRONIC PANCREATITIS. *(Fr.)* Callet, B.; Bernades, G.; Dupuy, R. (Beaujon Hosp., Clichy, France). *Arch. Fr. Mal. App. Dig.* 63(8):625-630, 1974.

Cholelithiasis was diagnosed in 17 (14 men and three women) of 101 patients with chronic pancreatitis. Chronic alcoholism (100 ml/day of pure alcohol for at least 2 yr) was present in 80 cases (75 men and five women) and pancreatic calcifications were detected in 71 cases. The diagnosis of cholelithiasis was made before that of chronic pancreatitis in three cases, simultaneously in ten cases and afterwards in four. No significant differences in sex, frequency of chronic alcoholism or acute pancreatitis, presence of pancreatic calcifications or associated cirrhosis or the course of chronic pancreatitis (regression, stabilization, exacerbation) were found between patients with chronic pancreatitis and those with chronic pancreatitis and cholelithiasis. Patients with cholelithiasis averaged 10.5 yr older at the onset of symptoms of chronic pancreatitis and 7.6 yr older at the diagnosis of chronic pancreatitis. Of the patients with cholelithiasis, 16 underwent surgery for cholecystitis (nine cases), prolonged cholestatic jaundice (six cases), or pancreatectomy (one case); the remaining patient died of cirrhosis and cholelithiasis was found at autopsy. It is concluded that the association between chronic pancreatitis and cholelithiasis is fortuitous and that cholelithiasis appears to play no role in the pathogenesis of chronic pancreatitis.

6378 TREATMENT OF PATIENTS WITH CHOLESTEROL GALLSTONES WITH CHENODEOXYCHOLIC ACID: CHANGES IN BILE ACID METABOLISM. *(Ger.)* Stiehl, A.; Raedsch, R.; Regula, M.; Kommerell, B. (Med. Clin., Univ. Heidelberg, Germany). *Inn. Med.* 2(1):13-18, 1975.

Chenodeoxycholic acid (CDC, 1.5 g/day) was used to treat ten patients, aged 35-74 yr, with cholesterol gallstones. Bile acid determinations were performed on fasting blood samples, 24-hr urine specimens and bile before and at various periods after institution of CDC therapy. CDC produced marked changes in the bile salt pool: significant increases occurred in CDC, ursodeoxycholic and lithocholic acids. While less than 5% of CDC, ursodeoxycholic, cholic and deoxycholic acids were present as the sulfate ester after administration of CDC, 67.4% of lithocholic acid was found in this form. Administration of CDC almost tripled serum concentrations of nonsulfated CDC and lithocholic acid, while serum levels of the sulfate esters of these acids remained almost the same. Only traces of ursodeoxycholic, deoxycholic, and cholic acids were found in the serum of these patients after CDC therapy. Administration of CDC significantly increased urinary excretion of the sulfate esters of CDC and lithocholic acids. In patients receiving CDC, serum concentrations of nonsulfated CDC, but not those of nonsulfated lithocholic acid, were increased by a test meal. These findings suggest that increases in serum bile acid concentrations may be due to a spillover of the bile acid pool. This spillover is increased by administration of a test meal. Since animal experiments have demonstrated that the sulfate ester of lithocholic acid is much less hepatotoxic than the acid itself, extensive sulfation of lithocholic acid in patients given CDC may be a mechanism protecting against liver injury.

6379 THERAPEUTIC PROPERTIES OF A CHOLERETIC AGENT, FLASTAPIOL, IN BILIARY DISEASE IN CHILDREN. *(Ukr.)* Shmorhun, S. S.; Zinchenko, T. V.; Maksiutina, N. P. (Kiev Inst. Postgrad. Med., USSR). *Pediatr. Akush. Ginekol.* 36(2):22-24, 1974.

Therapeutic application of flastapiol, a drug prepared from extracts of parsley fruit *(Petroselinum sativum)*, *Stachys recta* (a hedge-like perennial native to southeastern Europe and the Caucasus), sandwort (*Helichrysum arenarium*), pectin and sugar showed very good results in curing biliary disease in children. The drug was administered to patients aged 5-15 yr three times a day in the following doses: children 5-8, 0.5 g; children 9-12, 0.75 g; and children over 13, 1.0 g. Treatment lasted 14-26 days. Subjected to the study were 60 children: 36 with angiocholecystitis; 10 with cholecystitis; and 14 with angiocholitis. Treatment with flastapiol showed in general quick subjective improvement in the health of patients, a decrease in swelling of the inflamed liver, and the disappearence of abdominal pains. In addition, flastapiol showed very strong curing action on inflamed bile ducts. After treatment, the signs of inflammation in the duodenum of 51 (out of 60) children disappeared completely; five showed strong suppression; four, no effect. As a result of this clinical study, authors strongly recommend flastapiol, a drug with stimulating property on bile secretion of liver, as well as with spasmolytic, diuretic, and detoxicating properties for the treatment of biliary disease in children.

6380 KINETICS OF TRIGLYCERIDE METABOLISM DURING CHENODEOXYCHOLIC ACID THERAPY. *(Ger.)* Begemann, F. (Eppendorf Univ. Hosp., Hamburg, Germany). *Z. Gastroenterol.* 13:314-317, 1975.

By using Farquhar's two-compartment model and precursor labeling with ^{14}C-palmitic acid, administered i.v., triglyceride kinetics were studied in five patients with cholelithiasis who were receiving 10-15 mg/kg of chenodeoxycholic acid. After 8 weeks of therapy, plasma triglyceride levels had decreased 10 to 20%. Studies performed before and after 8 weeks of drug therapy in three cases showed that decreased plasma triglyceride levels were associated with a decrease in the rate of plasma triglyceride turnover. This demonstrated that decreased plasma triglycerides were due primarily to a decrease in the rate at which endogenous triglycerides entered the plasma and not to an increase in peripheral catabolism. These findings could either be attributed to a decrease in endogenous triglyceride synthesis or to a decrease in plasma secretion of these triglycerides. Since the supply of triglyceride for plasma secretion increased in two cases and decreased in one, the results were inconclusive.

6381 CONGENITAL BILIARY ATRESIA. *(Ger.)* Kleinteich, B.; Schickedanz, H. (Surg. Clin., Friedrich Schiller Univ., Jena, Germany). *Wiss. Z. Friedrich Schiller Univ. Jena, Math. Naturwiss. Reihe* 23(3/4):609-623, 1974.

In a 12-yr period, atresia of the extrahepatic biliary tract was successfully treated in only one of ten infants (six girls and four boys). Of these patients, seven had no symptoms for the first 2 weeks after birth, although acholic stools were present from birth in nine cases. The single exception was a girl whose stools became acholic 3 weeks after birth, but regained their normal color after 1 week. Laparotomy, performed at 6 weeks of age, showed atresia of the intrahepatic bile ducts and of the hepatic duct. However, since it was possible to pass a tube through the common bile duct, and contrast medium (injected into the gallbladder) passed through the bile duct and into the duodenum, accessory bile ducts were probably present. All patients eventually developed jaundice and hepatomegaly; only two had splenomegaly. Serum bilirubin levels, which ranged from 7.8 to 15.8 mg/100 ml, were not correlated with age, and direct bilirubin values were higher than indirect values in all cases. While no urobilinogen was detected in the urine in any of these infants, bilirubin was present in six cases. The WBC was increased (10,000 to 17,000 WBC/mm^3) in all cases. None of the patients had any evidence of syphilis, toxoplasmosis or listeriosis. At laparotomy, only two patients had operable forms, and choledochoduodenostomies were performed. One of these operated patients died 2 weeks after surgery, with liver failure due to progressive biliary cirrhosis. The other operated infant proved to have atresia of only a short segment of the common bile duct anterior to the papilla of Vater; all other extrahepatic bile ducts were normal. Bile and many small, pigmented gallstones were present in the common bile duct. The patient was discharged and remained in good condition 12 yr after surgery. Autopsies, performed in seven of the nine infants, showed that the cause of death was cirrhosis. Associated gastrointestinal anomalies were found in three patients, which consisted of hepatic, pancreatic, and intestinal malformations (one case), ileal stenosis (one case) and cystic fibrosis (one case).

6382 EMPHYSEMATOUS CHOLECYSTITIS: REPORT OF TWO CASES. *(Fr.)* Samson, E.; Kibrite, A.; Roy, J. P. (L'Hôtel-Dieu de Quebec Hosp., Quebec City, Canada). *Can. J. Surg.* 18(2):192-196, 1975.

Two new cases of emphysematous cholecystitis, a relatively rare disorder, are reported and compared to other cases from world literature. Both patients were male; one of them was diabetic. Both men presented with abdominal pain of sudden onset, which was localized around the right hypochondrium, extending to the shoulders in one case and to the paraumbilical region in the other. Simple radiography of the abdomen demonstrated a gas-filled gallbladder which led to the diagnosis of emphysematous cholecystitis. Surgery, supported by antibiotics, revealed gas, stones and distended gallbladder in both patients, with necrosis of the vesicular wall in one, and onset of necrosis in the other. It is felt that none of the etiological theories proposed thus far can be accepted or rejected, due to very limited experience with emphysematous cholecystitis.

6383 EFFECTIVENESS AND LIMITATIONS OF INTRAVENOUS CHOLANGIOGRAPHY IN PANCREATIC DIAGNOSIS. *(Ger.)* Frommhold, W.; Frommhold, H. (Inst. Med. Radiol., Univ. Tubingen, Germany). *Leber Magen Darm* 4(5):212-221, 1974.

Intravenous cholangiography can be used only to a very limited extent in the differential diagnosis of pancreatic disease because: (1) pancreatic diseases produce nonspecific morphological changes in the extrahepatic biliary tract and (2) good visualization of the common bile duct, which is necessary for the detection of these changes, cannot be obtained in 10 to 15% of i.v. cholangiograms. However, better analyses of the distal common bile duct can be obtained by infusing, rather than injecting, contrast medium and by using tomography and zonography. Zonography is also useful in detecting stones that do not produce shadows with conventional radiological techniques. Changes in the distal common bile duct are not necessarily specific for mechanical obstructions of the papilla since they can also be seen in diseases involving the head of the pancreas. Similarly, changes produced in the common bile duct by chronic pancreatitis or fibrosis of the head of the pancreas are also observed in pancreatic tumors or cholangiocarcinomas. However, these changes, when associated with pancreatic calcifications, suggest a diagnosis of chronic pancreatitis. Secondary changes in the common bile duct caused by acute pancreatitis are difficult to interpret. Pancreatic pseudocysts and retention cysts in the head of the pancreas occasionally displace and compress the common bile duct. Carcinomas of the head of the pan-

creas often displace the intrapancreatic part of the common bile duct in a lateral-convex manner, but similar changes also occur in pancreatitis. If a malignant tumor in the head of the pancreas produces stenosis in the upper biliary tract, cholangiocarcinomas and inflammatory processes, e.g. stenotic cholangiolitis, must be considered in a differential diagnosis. In low stenosis of the common bile duct with prestenotic dilatation of the proximal part, both papillary carcinoma and cicatricial, inflammatory changes in the head of the pancreas are possible diagnoses. In a few cases, i.v. cholangiography, in association with other conventional radiological techniques, e.g. barium swallow or hypotonic duodenography, may be helpful in the differential diagnosis of pancreatic disease, but in most cases it can only be regarded as a tool useful in preliminary diagnosis.

6384 GALLSTONES IN CHILDHOOD. *(Ger.)* Kock, H. J.; Reuter, G.; Noack, R. (Clin. Pediatr. Surg., Berlin, Germany). *Kinderaerztl. Prax.* 42(11):481-486, 1974.

Case reports are presented for six children (five girls and one boy, aged 9-14 yr) who were treated for cholelithiasis in a 17-yr period. Only two of these patients developed jaundice; one also had hepatomegaly and signs of parenchymal liver damage. Radiological findings were negative in three of the five children examined. Cholecystolithiasis alone was diagnosed in two cases. In one case it was associated with stenosis of the papilla of Vater and dilatation of the cystic and common bile ducts and in three cases with choledocholithiasis. In addition to cholecystectomy, two patients underwent choledochoduodenostomy and one underwent revision of the common bile duct. Gallstones, analyzed in five cases, consisted of cholesterol alone in four and mixed cholesterol-calcium oxalate stones in one.

6385 THE DISSOLUTION OF GALLSTONES WITH AND WITHOUT CHENODEOXYCHOLIC ACID. *(Ger.)* Mockel, G. (Hosp. TABEA, Hamburg-Blankenese, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:451-455, 1974.

For 9 months, a group of 25 patients (eight men and 17 women, aged 26-70 yr) with cholelithiasis were treated with chenodeoxycholic acid (average daily dose of 1 g). The following were considered contraindications for chenodeoxycholic acid therapy: (1) liver disease, wasting diseases, gastrointestinal disease with diarrhea, hematological disease, and impaired renal function; (2) pregnancy; and (3) patients in whom surgery was indicated. Almost no side effects occurred when this dose was used, while at higher dosages a few patients developed diarrhea. Diarrhea was alleviated by reducing the dosage to 0.75 to 1.0 g. Routine transaminase determinations showed no changes. Multiple gallstones, about the size of grains of rice, had disappeared from a 64-yr-old patient after 6 months of treatment with chenodeoxycholic acid. A 58-yr-old woman, who originally had six gallstones the size of cherries, had only two on a radiological examination performed after 8 months of treatment. In both cases, the gallstones had been dissolved without the development of any symptoms. Because gallstones tend to recur after they have dissolved, these patients continued to receive chenodeoxycholic acid.

6386 INCREASING PRODUCTION OF THE SULFATE ESTER OF LITHOCHOLIC ACID DURING CHENODEOXYCHOLIC ACID TREATMENT. *(Ger.)* Raedsch, R.; Stiehl, A. (Med. Clin., Univ. Heidelberg, Germany). *Verh. Dtsch. Ges. Inn Med.* 80:447-448, 1974.

Since the concentrations of chenodeoxycholic acid (CDCA) and lithocholic acid (LCA) increase in the bile of patients undergoing treatment of cholelithiasis with CDCA and the concentrations of these bile acids might be hepatotoxic, studies were performed to determine whether the relative percentages of their sulfate esters increase and what serum and urinary bile acid concentrations are during CDCA therapy. In patients with cholesterol gallstones given 1.5 g/day of CDCA p.o., the nonsulfated fraction of serum CDCA increased significantly from 0.9 to 2.7 μg/ml, while only a slight increase occurred in the sulfated fraction of serum CDCA. The serum concentration of nonsulfated LCA increased significantly from 0.2 to 0.7 μg/ml, and the serum concentration of LCA sulfate increased significantly from 0.1 to 0.5 μg/ml. Since bile acid concentrations in the serum are subject to considerable circadian variations, their urinary excretion was measured in 24-hr urine samples. Little increase occurred in the urinary excretion of nonsulfated CDCA, but excretion of the sulfate ester of this bile acid increased significantly from 0.3 to 0.5 mg/24 hr. While urinary excretion of nonsulfated LCA underwent no appreciable change, the excretion of LCA sulfate increased from 0.4 to 0.6 mg/24 hr. Differences between blood concentrations and urinary excretion of the sulfate esters during CDCA therapy are attributed to their greater glomerular filtration or lower reabsorption in the proximal tubules than nonsulfated bile acids. Before and during CDCA therapy, the relative percentage of the sulfate ester of CDCA in the bile was 0.7%, while the relative percentage of the sulfate ester of LCA increased significantly from 3.8 to 37.7% after 8 weeks of therapy. Accumulation of LCA in the body is prevented by increased synthesis of its sulfate ester since the reabsorption of the sulfate ester is decreased in the intestine and its renal excretion is increased.

6387 VARIATIONS IN TOTAL BILE ACIDS AND THE ACTIVITIES OF SOME ENZYMES USED AS INDICATORS OF CHOLESTASIS IN THE BILE OF PATIENTS AFTER SURGERY. *(Ger.)* Massarrat, S.; Arendt, D.; Kordes, B.; Bauer, E.; Tucke, M. (Med. Clin., Univ. Marburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:530-533, 1974.

Starting 3 days after surgery, the rate of bile secretion was measured, and determinations of total bile acids and enzymes used as indicators of cholestasis (alkaline phosphatase, γ-glutamyltranspeptidase, and leucine aminopeptidase) were performed on bile samples taken from T-tubes in 11 patients with jaundice caused by cholelithiasis and in ten anicteric pa-

tients with cholelithiasis. Bile samples were taken at 7 and 11 a.m. and at 4 and 9 p.m. In both groups of patients, very close correlations were found between the secretion of bile acids/min and alkaline phosphatase activity/min and between the secretion of bile acids/min and the volume of bile secreted/min. Close correlations were also found between alkaline phosphatase and γ-glutamyltranspeptidase activities in both groups of patients. When enterohepatic circulation was reestablished by raising the T-tube, bile acid concentrations increased; bile acid concentrations and alkaline phosphatase and γ-glutamyltranspeptidase activities showed circadian variations. Bile acid concentrations and enzyme activities were highest at 7 and 11 a.m., decreased after lunch and, after a dinner at 6 p.m., increased again at 9 p.m. This suggests that bile acid concentrations and enzyme activities are subject to a circadian rhythm, but the effect of food ingestion cannot be ruled out. Changes in the alkaline phosphatase activity in the bile proved to be a better indication of changes in concentration than did the bile acid concentration.

6388 TURNOVER OF PLASMA CHOLESTEROL IN PATIENTS WITH CHOLESTEROL GALLSTONES. *(E.)* Pedersen, L.; Arnfred, T.; Thaysen, E. H. (Dep. Med., Alborg Hosp. North, Denmark). *Acta Med. Scand.* 197(5):421-425, 1975.

Some kinetic parameters of cholesterol synthesis were measured in eight Caucasian patients with radiolucent gallstones (in well functioning gallbladders) and in two healthy controls. 1-α-2-α-^{3}H-cholesterol in ethanol solution (2 to 3 ml) was administered i.v. to all subjects. In three patients, the half-life of the radiocholesterol in the circulation was 10 min and the distribution of radioactivity in the lipoproteins was similar to that of cholesterol in healthy controls. Fasting plasma samples were obtained once or twice weekly from all subjects for 10 weeks following injection of the tracer, and the plasma cholesterol and plasma triglyceride levels were determined. In each subject some kinetic parameters were also calculated, including the input into and size of the cholesterol pool. The results were compared with those from 16 controls in a previous study. The statistical analysis failed to demonstrate significant differences between the gallstone patients and compiled controls in any of the parameters studied. The results indicate that patients with cholesterol gallstones have normal rates of input of cholesterol into the rapidly exchangeable cholesterol mass. It is suggested that the absorption of exogenous cholesterol may be decreased in gallstone patients.

6389 ADENOMYOMATOSIS OF THE GALLBLADDER. *(E.)* Ram, M. D.; Midha, D. (Huron Road Hosp., Cleveland, Ohio). *Surgery* 78(2):224-229, 1975.

Adenomyomatosis of the gallbladder in 27 patients was reviewed to investigate the nature and treatment of the disease. Nine were diagnosed directly by radiography, 14 had gallstones, three had a non-visualized gallbladder, and one case was discovered on surgery. Ages ranged from 21-72 yr with the highest incidence in the sixth and seventh decades of life. Of the 27, 25 were white and 17 were female. Clinical features included pain in the right hypochondrium, dyspepsia and fatty food intolerance. Duration of symptoms ranged from 3 months to 7 yr. Cholecystectomy was performed in each case. Two patients died of unrelated causes; 25 were asymptomatic six months later, and 20 who were followed up to a maximum of 7 yr remained asymptomatic. Radiology showed morphological changes best after a fatty meal. Contrast filled the dilated elements in the wall in the form of a saw-tooth edge along the margin. In the segmental variety of the disease, concentric narrowing was seen. Pathology showed gross thickening of the wall, confined to a definite area in the segmental and localized varieties. Histologically there was hyperplasia of the epithelial and muscular elements, intramural diverticulosis extending into the subserosal plane, and thickening of cell layers. It is concluded that the disease is probably due to a combination of proliferation and degeneration associated with functional dyskinesia, and is curable by cholecystectomy.

6390 EARLY FORMATION OF GALLSTONES FOLLOWING JEJUNOILEAL BYPASS FOR TREATMENT OF MORBID OBESITY. *(E.)* Neshat, A. A.; Flye, M. W. (Duke Univ. Med. Cent., Durham, N.C.). *Am. Surg.* 41(8): 486-491, 1975.

A case of cholelithiasis and acute cholecystitis immediately following a jejunoileal bypass is presented. A 30-yr-old male presented with morbid obesity. All laboratory studies were within normal limits, except alkaline phosphatase levels 90 mμ/ml. Jejunoileal bypass was performed, at which time the gallbladder was found to be grossly normal without palpable calculi. Liver biopsy showed minimal fatty changes. He was discharged 2 weeks after surgery, but presented 6 days later with epigastric pain, nausea, and vomiting. He responded to bed rest, propantheline bromide and bland diet, and was discharged after 3 days. Five weeks after surgery he presented with abdominal pain and fatty food intolerance. Oral cholecystography revealed multiple small stones in the gallbladder. He responded to conservative treatment. Two weeks later he presented with right upper quadrant pain, nausea and vomiting. Laboratory tests showed uric acid (7.1 mg%), alkaline phosphatase (175 mμ/ml), and serum iron (62 mg%). Cholecystectomy was performed and multiple small, mushy gallstones were found. Analysis showed them to be composed of cholesterol and calcium bilirubinate. Liver biopsy showed severe fatty changes. It is suggested that typical postoperative metabolic changes, mostly of fat and protein malabsorption, potassium and calcium loss from diarrhea, and bile stasis from obesity affected the cholesterol to bile salt plus lecithin ratio in the bile, resulting in stone formation.

6391 CLINICOPATHOLOGICAL STUDIES ON THE RELATIONSHIP BETWEEN CHOLELITHIASIS AND CHRONIC PANCREATITIS. *(E.)* Sato, T.; Saitoh, Y.; Noto, N.; Matsuno, S.; Tokutake, K. (Tohoku Univ. Sch. Med., Sendai, Japan). *Tohoku J. Exp. Med.* 113(2):97-111, 1974.

To clarify the relation between cholelithiasis and chronic pancreatitis, biopsies of the pancreas were obtained at laparotomy from 151 patients (69 men and 82 women, aged < 19 to > 80 yr) with cholelithiasis, and the secretin test of pancreatic exocrine function performed on 119 of these patients. Histologic findings were compared with those obtained in 31 patients with gastric cancer. Pancreatic exocrine function was normal in 89 (74.8%) of the 119 patients who underwent the secretin test, slightly disturbed in 25 (21.0%), moderately disturbed in 4 (3.4%), and markedly disturbed in 1 (0.8%). Of the 30 patients with disturbance, 17 had reduced maximum bicarbonate concentration, 10 had reduced volume of duodenal aspirate, and 9 had reduced total amylase output. Moderate or marked disturbance of pancreatic exocrine function was significantly more frequent in patients with bacteria in bile, dilation of the bile duct, and hypercholesterolemia. Results of the secretin test showed a significant correlation with the frequency and grade of histologic changes in the pancreas: while 75-78% of the patients with a normal test showed no histologic change, 4 of 5 with a moderate or marked disturbance in the secretin test had marked histologic changes. However, no significant difference in grade and frequency of histological change in the pancreas was demonstrated between patients with cholelithiasis and controls. In addition, histologic findings did not correlate with location and type of gallstones or with duration of symptoms. Only 5 (3%) of the 151 patients with cholelithiasis had chronic pancreatitis. The results support previous findings that the frequency of pancreatitis is not high in patients with cholethiasis even though there should be common constitutional factors existing between cholelithiasis and chronic pancreatitis.

6392 ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY FOLLOWING BILIARY SURGERY. *(Ger.)* Stadelmann, O.; Kaufer, C.; Bogusch, G.; Miederer, S. E.; Sobbe, A.; Slotter, H.; Loffler, A. (Med. Polyclin., Univ. Bonn, Germany). *Leber Magen Darm* 4(5):201-211, 1974.

Endoscopic retrograde cholangiopancreatography (ERCP) was performed on 113 patients who had undergone biliary surgery and had postoperative upper abdominal discomfort, signs of cholestasis, or laboratory findings suggestive of biliary obstruction. ERCP was used only if i.v. cholangiography could not be performed or gave no clearcut diagnostic information. In cases in which at least one system of ducts was visualized, the biliary tract was visualized in 80% and the pancreatic tract in only 66%. In 52 cases, both pancreatic and biliary ducts were visualized; in 38, only the biliary ducts; and in 23, only the pancreatic ducts. Pathological changes were detected in 72 cases (64%); in the biliary tract in 61 of 90 cholangiograms and in 22 of the 75 pancreatograms; in 11 cases both systems were affected. The most commonly encountered changes in the biliary tract were papillary stenosis (39 cases), gallstones (11), and stenosis associated with gallstones (9). Hydatid disease of the liver was associated with prepapillary stenosis in one case, and Caroli's syndrome was associated with choledocholithiasis in another. Pancreatic disease, diagnosed in 15 cases, consisted of chronic pancreatitis in 11 patients and pancreatic carcinomas in four. The diameters of the common bile duct and hepatic ducts increased with age and in patients who had undergone cholecystectomy, providing that no pathological changes, particularly obstructions, were present. Some patients who had undergone choledochoduodenostomy developed blind loop syndrome and papillary stenosis. It is recommended that this procedure be combined with papillotomy.

6393 CHANGES IN BILE ACID METABOLISM DURING TREATMENT WITH CHENODEOXYCHOLIC ACID. *(Ger.)* Stiehl, A. (Med. Clin., Univ. Heidelberg, Germany). *Z. Gastroenterol.* 13(suppl.):300-301, 1975.

In 10 patients with cholelithiasis, serum levels of nonsulfated chenodeoxycholic acid (CDCA) increased significantly from 0.9 µg/ml to 2.7 µg/ml during treatment with 1.5 g/day CDCA; CDCA sulfate levels remained essentially the same. After a test meal, serum CDCA values increased still further to 4.6 µg/ml, while CDCA sulfate only increased to 0.5 µg/ml. Serum lithocholic acid concentrations also increased significantly from 0.2 µg/ml before treatment to 0.7 µg/ml during treatment; the sulfate ester level of this acid also increased from 0.1 to 0.5 µg/ml. No appreciable changes occurred in levels of serum lithocholic acid or its sulfate after a test meal. Urinary excretion of CDCA increased only slightly (from 0.5 to 0.6 mg/24 hr) during CDCA treatment, while urinary excretion of its sulfate ester increased significantly (from 0.2 to 0.5 mg/24 hr). Similar results were obtained for urinary excretion of lithocholic acid and its sulfates. Bile acid determinations performed on bile from these patients revealed that during CDCA therapy the relative percentage of CDCA increased from 47% to 72%; ursodeoxycholic acid, from 2% to 17%; and lithocholic acid, from 1% to 3%. The relative percentage of cholic acid in their bile decreased from 41% to 4%, and that of deoxycholic acid, from 9% to 3%. All of these changes were significant. Before CDCA therapy, only 22.5% of lithocholic acid in the bile was present as the sulfate ester; during therapy this increased steadily to 67.5%. Since lithocholic acid is hepatotoxic, it is suggested that formation of its sulfate ester is a protective mechanism which leads to its rapid detoxification and excretion.

6394 SCANNING ELECTRON MICROSCOPE STUDIES OF THE MUCOSAL SURFACE IN THE GALLBLADDER IN CHOLECYSTITIS AND CHOLELITHIASIS. *(Ger.)* Wagner, R.; Krieg, H. (Pathol. Inst., Johannes Gutenberg Univ., Mainz, Germany). *Z. Gastroenterol.* 13(2):348-355, 1975.

To determine the extent to which structural changes in the gallbladder mucosa contribute to lithogenesis, the mucosal surface of gallbladders removed from 11 women with cholecystitis was examined under the scanning electron microscope. Cholecystitis was associated with a polyp in one case and with cholelithiasis in 10. In the gallbladder from the patient with the polyp, the mucosal surface contained folds of various widths and depths which were located so close to one another that the crypts could not be seen; a few adhesions were observed between adjacent folds. The epithelium consisted of polygonal cells of various sizes. Microvilli of various heights were detected on the apical parts of these cells. In a few places, very small processes, called antennulae, projected from the protuberances on the distal ends of the microvilli. In gallbladders from seven patients with chronic cholecystitis, the mucosal relief was flattened so that the crypts could be seen. While some deep crypts measured 5-80 μ in diameter, shallow crypts were considerably wider. In addition to these changes, the mucosal surface in two patients with acute cholecystitis contained epithelial ulcerations containing fibrils of collagen and fibrin. There were many loose cells, mostly RBC, among the fibrils; some cells were deformed by the fibrin. Intracellular spaces were increased in width and some contained microvilli of different heights. Microliths were detected near isolated necrotic cells and the flattened epithelium, while larger stones were present in the crypts. In addition to a few pigment stones, stones of pure cholesterol were found. Older solitary stones of cholesterol were joined to form thick plates, while multiple, recently formed cholesterol stones consisted of thin, loosely joined plates. It is suggested that the microliths might produce cellular necrosis, resulting in an increase in the size of the stones and in the width of the crypts and causing flattening of the mucosal relief. However, it is not known whether this process is produced by cholecystitis or whether inflammation results from stone formation.

6395 SPONTANEOUS ENTEROBILIARY FISTULAS: SURGICAL EXPLORATION AND SURGICAL POLICY. EIGHT CASES. *(Fr.)* Chartier, P. (no affil.) *Ouest Med.* 27(18):1673-1682, 1974.

To illustrate the causes of internal enterobiliary fistulas and surgical procedures for treating them, case reports are presented for eight patients (five women and three men, aged 41-82 yr). These patients constituted 4.3% of all 189 patients who underwent biliary surgery. A 63-yr-old man with a cholecystoduodenal fistula and a gallstone obstructing his duodenum was treated by cholecystectomy and duodenal suture, as was a 68-yr-old woman who had a cholecystoduodenal fistula associated with a sclerotic, atrophic gallbladder. A cholecystoduodenal fistula associated with a duodenal ulcer in a 41-yr-old man was treated by a Péan-type gastrectomy, cholecystectomy, and insertion of a Kehr drain. A 66-yr-old man with a choledochoduodenal fistula, who had undergone two operations for peptic ulcer complications, was treated by gastrectomy, a Y-shaped enterobiliary anastomosis, and Finsterer's gastrojejunostomy. A cholecystocholedochal fistula, associated with gallstones in the gallbladder, common bile duct, and liver in a 82-yr-old woman, was treated by cholecystectomy, choledochotomy, and choledochoduodenal anastomosis. A 79-yr-old woman who had a duodenocholedochal fistula, a duodenal diverticulum, cholangitis, and cancer with partial obstruction of the transverse colon underwent cholecystectomy, resection of the duodenal diverticulum, a Y-shaped enterobiliary anastomosis, and segmental colectomy. Fistulas between the gallbladder and hepatic duct in a 71-yr-old and a 69-yr-old woman, which were masked by cholelithiasis, were treated by cholecystectomy and a Y-shaped anastomosis between the hepatic duct and jejunum. All patients except the one with cancer of the colon survived. Five patients had no complications and were discharged from the hospital 10-21 days after surgery. One patient was given anticoagulants after she developed myocardial ischemia 48 hr after surgery, and three received antibiotics.

6396 CONGENITAL CYSTS OF THE INTRAHEPATIC BILIARY TRACT (CAROLI'S DISEASE). *(Ger.)* Siedek, M.; Kaufer, C.; Clemens, W.; Stadelmann, O.; Bucheler, E. (Surg. Clin., Univ. Bonn, Germany). *Leber Magen Darm* 4(5):242-246, 1974.

A 32-yr-old man, whose brother had chronic hepatitis, was hospitalized eight times in a 10-yr period for undiagnosed pain in the upper right abdomen. A cholecystectomy, performed for suspected cholecystitis, showed no evidence of stones in the gallbladder; a choledocholithotomy was performed at the same time. The biliary tract was not visualized radiologically. The following year, the patient was hospitalized for cholangitis and pancreatitis and was treated with antibiotics. The next year, he was hospitalized again for colic-like pain in the upper right abdomen. An infusion cholangiogram performed with tomography showed weak contrasting of dilated bile ducts in the liver and a dilated common bile duct with prepapillary stenosis and a suggestion of gallstones. No evidence of tumors or echinococcal cysts was found by radiological examination of the hepatic duct or by splenoportography. Some branches of the hepatic artery and portal vein were compressed, and the configuration of the left branch of the portal vein was atypical. Choledocholithotomy and transduodenal papillotomy were performed after removal of four prepapillary stones. A diagnosis of Caroli's disease was established by transpapillary visualization of multiple cysts in the intrahepatic bile ducts. Histological examination of a liver biopsy showed fibrosis associated with pericholangitis. *Klebsiella, Pseudomonas,* and enterococci were cultured from the bile. The patient was given antibiotics after surgery and recovered uneventfully.

6397 CARCINOMA OF THE DUODENAL PAPILLA: BYPASS, WHIPPLE'S PROCEDURE, OR PAPILLECTOMY? *(Ger.)* Enderlin, F. (Cantonal Hosp., Basel, Switzerland). *Schweiz. Med. Wochenschr.* 105(18):592-595, 1975.

In a 20-yr period, surgery was performed on 41 patients, aged 47 to 80 yr (mean, 62 yr) with carcinomas of the duodenal papilla. The procedure employed was palliative in 16 cases (12 enterobiliary anastomoses and four biliary fistulas), although only three of these 16 patients had confirmed metastases. The surgical mortality was 30%, and the average survival time was only a few months; 2/3 of the patients died within 1 yr. Four of the 16 had undergone cholecystectomy 1 to 3 months before tumor surgery. Whipple's procedure was performed on 17 patients, four of whom had been previously cholecystectomized; five had confirmed lymph node metastases. In noncholecystectomized patients, the gallbladder was removed as a site of potential inflammation, as was the distal part of the stomach, to prevent the development of ulcers. Of these 17 patients, two underwent surgery within the last month and three are alive 2.7, 5, and 22 yr after surgery. Tumors or metastases resulted in death in 12 cases, but eight of these survived more than 1 yr and three survived more than 2 yr. Papillectomies were performed on three patients with localized tumors and on five elderly patients. Two patients are apparently tumor-free after 4 yr, but two others have died of recurrences.

6398 ULTRASOUND TOMOGRAPHY: A SAFE METHOD FOR THE DIAGNOSIS OF CONGENITAL CYSTS OF THE COMMON BILE DUCT. *(Ger.)* Weitzel, D.; Beck, J. D. (Pediatr. Clin., Univ. Mainz, Germany). *Klin. Paediatr.* 186(5):460-464, 1974.

A 5-yr-old girl was suspected of having chronic aggressive hepatitis because of a history, dating back to infancy, of episodes of diarrhea and vomiting which recurred at 4 to 10 week intervals and were accompanied by colic-like pain, slimy, light yellow stools and dark urine. She failed to respond to diet therapy, fructose infusions, and fluocortolone administered over a 3-month period. At admission she had jaundice, hepatomegaly, an increased RBC sedimentation rate, and increased WBC, SGOT, SGPT, bilirubin and α_2-globulin values. Indirect evidence of a space-occupying lesion in the pancreas or hepatic portal was obtained by a barium swallow examination, and a liver scan indicated the lesion was located in the right lower lobe. Findings from celiac angiography were consistent with an avascular tumor, and larparoscopy revealed a full gallbladder, massive dilatation of the common bile duct, and an enlarged liver with gross signs of biliary cirrhosis. Findings obtained with ultrasound tomography B were interpreted as excessive dilatation of the bile duct, enlargement of the gallbladder, enlargement of the intrahepatic bile ducts, and diffuse connective tissue proliferation in the liver. At surgery, the common bile duct was about 12 to 15 cm long and 3-5 cm in diameter and, on gross examination, was closed near the papilla. When this bile duct was punctured, 140 ml of bile were obtained. A choledochoduodenostomy was performed and the patient recovered uneventfully. Pathological laboratory findings normalized within a few weeks. Examination of a biopsy taken from the liver at surgery showed biliary cirrhosis. Ultrasound tomography is recommended as the only technique for diagnosing biliary cysts since it shows the space-occupying lesion and its location directly and confirms the fluid nature of the cyst.

6399 PRIMARY CARCINOMA OF THE GALLBLADDER: REPORT ON 36 CASES. *(Ger.)* Von Schoning, G.; Anders, A.; Pludra, J. E. (Steglitz Clin., Free Univ., Berlin, Germany). *Chirurg* 46(1):40-46, 1975.

Primary carcinoma of the gallbladder was diagnosed in 36 of 1228 operations on the biliary tract performed in a 4-yr period (2.9%). These 36 patients consisted of 34 women and two men; all but one were over 50 yr old (mean age 69.2 yr). The most common symptom was pain, present in all but four of these patients; upper abdominal tumors or hepatomegaly were found on palpation in 25 cases. None of the laboratory tests performed (alkaline phosphatase, transaminase, hemoglobin, SBC, RBC sedimentation rate and bilirubin) were diagnostic for carcinoma of the gallbladder. Cholangiography was of little value, because the biliary tract was not visualized in jaundiced patients. Barium swallow only suggested the presence of tumors in the biliary tract if the tumor compressed the duodenum and stomach or if a biliary fistula was present. The diagnosis was not established at surgery in six cases, although an experienced pathologist was able to diagnose carcinoma in three of these cases by gross examination of the tissues removed at surgery. This suggests that frozen sections should be examined during surgery. Histological examinations showed that 27 of the 36 patients had adenocarcinomas; these included seven solid undifferentiated and seven mucus-secreting adenocarcinomas. Of three squamous cell carcinomas, two were keratinized. Undifferentiated solid carcinomas were found in three patients, and no histological information was available for three others. Although surgery is generally palliative (carcinomas of the gallbladder soon invade the liver and then the regional lymph nodes), five patients were asymptomatic for more than 12 months after surgery; one patient had a localized carcinoma of the gallbladder and it was possible to remove regional metastases in the other four cases.

6400 ENDOSCOPIC PAPILLOTOMY. EXPERIMENTAL STUDIES AND INITIAL CLINICAL EXPERIENCE. *(E.)* Koch, H.; Classen, M.; Schaffner, O.; Demling, L. (Med. Dep., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Scand. J. Gastroenterol.* 10(4):441-444, 1975.

Experimental studies and initial clinical experience in the use of a high-frequency diathermy probe to perform endoscopic papillotomies are reported. Satisfactory papillotomies were performed on 11 dogs, and on the basis of these results endoscopic papillotomies were carried out in 23 patients. Indications for endoscopic papillotomy included common bile duct calculi in 11 high-risk patients; common bile duct calculi overlooked during bile duct surgery in six patients; recurrent calculi after previous cholecystectomy in three cases; and circumscribed papillary stenosis in three patients. For endoscopic papillotomy, the following procedure was adopted: retrograde con-

trast medium visualization of the common bile duct, possibly also of the main pancreatic duct; introduction of the papillotomy probe; accurate radiological check of the position of the papillotomy probe; and papillotomy. Papillotomy was successful in all 23 cases. At the endoscopic and radiologic follow-up examination carried out three to five days after papilotomy, no further stones were demonstrable in the bile duct in nine cases, indicating they had been passed spontaneously. In ten cases, the calculi had to be extracted using the Dormia basket or a wire loop. One patient had an attack of pancreatitis and the common bile duct had to be removed surgically. Further complications, in particular the occurrence of massive postoperative bleeding, were not observed. The clinical results show that endoscopic papillotomy carried out with the aid of a high-frequency diathermy probe is feasible in human patients and makes it possible to remove calculi in the common bile duct without opening the abdomen.

6401 DRUG ABSORPTION IN PATIENTS WITH T-TUBE AFTER CHOLECYSTECTOMY. *(E.)* Aukee, S.; Venho, V. M. K.; Jussila, J.; Karjalainen, P. (Univ. Cent. Hosp., Kuopio, Finland). *Ann. Clin. Res.* 7(1):42-46, 1975.

The influence of bile flow on gastrointestinal absorption of tetracycline, doxycycline, sulfamethazine, and cycloserine was studied in 13 volunteer patients with T-tubes in their main bile ducts following biliary surgery. Gastroduodenal tubes were installed 1 week after cholecystectomy, and hepatic function and bile flow were evaluated following the i.v. administration of 10 μCi of I^{131} rose-bengal. In one group of patients, 1 g of tetracycline hydrochloride aqueous suspension was then administered via the gastroduodenal tube; the tube was rinsed with water and 0.75 g of sulfamethazine was given as an aqueous solution. In the other group of patients, 0.2 g of doxycycline and 0.5 g of cycloserine were given p.o. In both groups, blood samples were taken before and at various intervals after drug administration. The experiments were repeated three days later, after the T-tube was closed. Liver function did not vary in either group whether the T-tube was open or closed. About 80% of the radioactive tracer was recovered from the bile when the T-tube was open, so most of the bile was diverted outside the intestinal tract. Up to 4 hr after administration, the serum tetracycline levels were lower when the T-tube was open than when it was closed. In one patient, the serum levels were decreased to such an extent that therapeutic serum tetracycline levels could not be achieved. The serum doxycycline levels were fairly high and bile flow did not have any effect on doxycycline absorption. The 24-hr biliary excretion of this drug was only about 15% of the urinary excretion. The absorption of sulfamethazine and cycloserine was not affected by the presence or absence of bile. The results indicate that enterohepatic circulation of doxycycline is not important for the maintenance of its serum levels, that intact bile flow is not essential for the absorption of sulfadimidine, and that with the doses currently used, therapeutic serum levels of tetracycline can be expected even in cases of diminished bile flow.

6402 THE REMOVAL OF RETAINED GALLSTONES FROM THE COMMON BILE DUCT: EXPERIENCE WITH SODIUM CHOLATE INFUSION AND THE BURHENNE CATHETER. *(E.)* Britton, D. C.; Gill, B. S.; Taylor, R. M. R.; James, O. (R. Victoria Infirm., Newcastle upon Tyne, England). *Br. J. Surg.* 62(7):520-523, 1975.

The use of cholic acid and the Burhenne catheter in the management of gallstones retained in the common bile duct following biliary surgery was studied in nine patients 3-16 weeks after cholecystectomy and exploration of the common bile duct. In four patients, an i.v. catheter was passed down through the T tube to the junction and a sodium cholate solution was infused at a rate of 30 ml/hr for up to 12 days. In two other subjects the T tube was removed and the Burhenne catheter was passed down the T tube tract and manipulated into the common bile duct; the stones were trapped in a Dormia basket passed through the catheter and removed. Three additional subjects received sodium cholate infusion followed by instrumentation with a Burhenne catheter. Liver function tests and estimations of serum electrolytes and serum amylase were performed daily and cholangiograms were performed at intervals of 4 days. Four of the seven patients treated with cholic acid infusion showed disappearance of the gallstones within 8-12 days. In another subject, one stone disappeared during infusion and a second stone was successfully removed with a Burhenne catheter; in one other patient, sodium cholate infusion was ineffective, but the stone was removed by instrumentation. Two attempts at using a Burhenne catheter alone failed as did one attempt at instrumentation following ineffective sodium cholate treatment. All patients treated with sodium cholate developed diarrhea, often quite severe, and became nauseated and anorexic and vomited intermittently. Biliary peritonitis occurred after use of a Burhenne catheter in one patient and after removal of the T tube in a patient treated with sodium cholate. The successful use of the Burhenne catheter appears to require considerable practice and expertise, and should perhaps be reserved for centers with particular interest in its use.

6403 BILIARY AND URINARY CALCULI: PATHOGENESIS FOLLOWING SMALL BOWEL BYPASS FOR OBESITY. *(E.)* Wise, L.; Stein, T. (Washington Univ. Sch. Med., St. Louis, Mo.). *Arch. Surg.* 110(8):1043-1047, 1975.

The pathogenesis of biliary and urinary calculi following small intestine bypass procedures was studied in 93 patients who had undergone jejunoileal bypass procedures for massive exogenous obesity. In each case, 35 cm of the proximal jejunum was anastomosed end-to-end with the terminal portion of the ileum. I.v. cholecystokinin was administered to the fasted subjects and bile was collected in a double-lumen gastroduodenal tube. (^{14}C)Cholate (10 μCi) and (^{14}C)chenodeoxycholate (10 μCi) were then administered i.v. and bile-rich duodenal fluid was collected 3, 24, 48, and 72 hr later. Bile salt synthesis and the bile salt glycine/taurine ratio were determined. Within a mean follow-up period of 17.6 mon, three of the 93 patients developed urinary calculi, four de-

veloped gallstones, and one developed both urinary calculi and gallstones. Four of the gallstone patients were asymptomatic and one complained of vague upper abdominal pain, while all of those with urinary stones were hospitalized with typical "ureteric colic." A significant postoperative increase in urinary oxalate excretion was correlated with a significant decrease in urinary volume. The 24-hr preoperative urinary oxalate excretion in the four patients who developed urinary calculi was less than 20 mg, while the postoperative values were 94, 107, 51, and 52 mg/24 hr. The stones in these cases were all calcium oxalate calculi and all patients passed them spontaneously or with the help of retrograde catheter manipulation. The rate of bile salt synthesis increased significantly from 1.6 g/day preoperatively to 4.9 g/day postoperatively, and the bile salt glycine-taurine ratio increased significantly from 4.6 to 6.8. It is suggested that the postbypass increase in the biliary glycine/taurine ratio, with its consequent decrease in the zeta potential of the bile micelles, was at least partially responsible for the increased incidence of cholelithiasis. The pathogenic basis for the increased incidence of urinary calculi was hyperoxaluria, which was probably related to the increased bile salt and glycine synthesis.

6404 THE RATE OF DRUG BIOTRANSFORMATION REACTIONS AND CELLULAR VIABILITY IN THE LIVERS OF PATIENTS WITH BILIARY DISEASES. *(E.)* Hietanen, E.; Auranen, A.; Savvakis, C. (Dep. Physiol. Surg., Univ. Turku, Finland). *Acta Hepatogastroenterol. (Stuttg.)* 22(3):170-175, 1975.

The determine how biliary diseases affect hepatic drug metabolism and cellular viability, the cytochrome P-450 concentration, rate of o-aminophenylglucuronide synthesis, and oxygen consumption were measured in liver slices from 62 female patients with uncomplicated cholelithiasis, chronic cholecystitis, choledocholithiasis without bile duct obstruction, or bile duct obstruction and jaundice. The hepatic cytochrome P-450 concentration was highest in the patients with uncomplicated cholelithiasis, slightly decreased in those with chronic cholecystitis, and decreased by about 20% (compared with the uncomplicated cholelithiasis patients) in the patients with bile duct stones without jaundice. The rate of o-aminophenylglucuronide synthesis was similar in the patients with gallstones, chronic cholecystitis, and choledocholithiasis without jaundice; this rate was significantly higher in the patients with obstructive icterus than in the patients with chronic cholecystitis and was significantly higher than in those with gallstones or choledocholithiasis without obstruction. The oxygen consumption by the liver slices from the patients with obstructive jaundice was higher than that by the liver slices from the other groups. The formation of carbon dioxide did not differ among the four groups. It is suggested that the enhanced rate of glycuronidation was caused by the endogenous induction of increased bilirubin levels or by the accumulation of bile acids in the liver cells. The data suggest that drug hydroxylation and glucuronidation activities are under separate control mechanisms.

6405 THE EFFECT OF SODIUM BENZOATE AND TAUROCHOLIC ACID FEEDING ON HUMAN BILE COMPOSITION. *(E.)* Dennis, C.; Gardner, B.; Patti, J.; Chenouda, M. (Downstate Med. Cent., State Univ. New York, Brooklyn, N. Y.). *Proc. Soc. Exp. Biol. Med.* 148(3):602-605, 1975.

The effect of sodium benzoate and taurocholic acid feeding on bile composition was investigated in four patients who had cholecystectomy for cholesterol gallstones and common bile duct exploration with the insertion of a Baldwin T-tube. To determine if the glycine/taurine conjugation ratio could be reversed under acute conditions, the patients were fed sodium benzoate alone (6 g/day) for two or three days and then in combination with taurocholic acid (3 g/day) for three days. No significant changes were noted in serum alkaline phosphatase, bilirubin or glutamic-oxalacetic transaminase levels during the experiment. The benzoate was incapable of significantly lowering the glycine conjugates in the bile. Taurocholic acid significantly increased the concentration of taurine conjugates and markedly altered the glycine/taurine conjugation ratio. The only consistent results were the increase in taurine conjugates after feeding taurocholic acid. Total bile acids were significantly increased in all patients with a marked fall in glycine/taurine conjugation ratio. A proportional increase in biliary phospholipids and cholesterol was also demonstrated. There was no consistent change in the ratio of cholate to chenodeoxycholate concentration in any of the patients. Three patients demonstrated a slight fall in bile acid excretion which returned to normal due to an increase of taurine conjugated bile acid excretion. The results show that the usual glycine/taurine conjugation ratio of 3 or 4:1 consistently found in humans is the result of a relatively low taurine pool available for conjugation of bile acids and not due to preferential conjugation with glycine.

6406 COMPLICATIONS AFTER BILE DUCT SURGERY. *(Ger.)* Wex, P.; Helmstaedt, E. R. (Surg. Clin., Bremen, Germany). *Med. Welt* 26(5):175-178, 1975.

6407 GALLBLADDER CARCINOMA. *(Ger.)* Moschinski, D.; Sailer, R.; Jacobs, G. (Surg. Clin., Univ. Dusseldorf, Germany). *Med. Welt* 26(5):171-175, 1975.

6408 DUODENUM-PAPILLA-PANCREAS: 1. MORPHOLOGY AND CLINICAL EXPERIENCE WITH DISEASES OF THE PAPILLAR REGION. *(Ger.)* Becker, V. (no affil.). *Fortschr. Med.* 93(5):193, 1975.

6409 INDICATIONS FOR TRANSDUODENAL SPHINCTEROTOMY. *(Ger.)* Grill, W. (no affil.). *Fortschr. Med.* 93(5):200-204, 1975.

6410 PATHOLOGICAL ANATOMY OF PAPILLOTOMY AND THE SITUATION AFTER PAPILLOTOMY. *(Ger.)* Fodisch, H. J. (no affil.). *Fortschr. Med.* 93(6):247-248, 1975.

6411 THE SIGNIFICANCE OF DISORDERS IN THE INTESTINAL FLORA IN BILIARY DISEASE IN CHILDREN. *(Rus.)* Tikhii, A. K.; Archakov, E. V.; Tikhaia, P. H.; Zaslavskaia, E. S. (Dnepropetrovsk Med. Inst., USSR). *Pediatriia* (3):40-41, 1975.

6412 BLOOD LEVELS OF TRACE ELEMENTS IN CHRONIC CHOLECYSTITIS IN CHILDREN. *(Rus.)* Siniavskaia, O. A.; Bakhrusheva, T. S. (Sverdlovsk Med. Inst., USSR). *Pediatriia* (3):44-45, 1975.

6413 THE ROLE OF BILE ACIDS IN THE FORMATION AND DISSOLUTION OF GALLSTONES. *(Ger.)* Weis, H. J. (Med. Clin., Univ. Mainz, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:338-343, 1974.

6414 RENAL FUNCTION FOLLOWING SURGERY FOR CHRONIC BILIARY DISEASE. *(Rus.)* Kubei, V. I. (Ternopol Med. Inst., USSR). *Klin. Khir.* (4):31-34, 1975.

6415 CONGENITAL DILATATION OF THE COMMON BILE DUCT. *(Rus.)* Zemskov, N. N.; Ablitsov, N. P. (Voroshilovgrad Med. Inst., USSR). *Klin. Khir.* (4):61-62, 1975.

6416 OBJECTIVE EVALUATION OF BILIARY DECOMPRESSION BY HEPATIC RHEOGRAPHY. *(Rus.)* Chekalin, A. P. (Donetsk Med. Inst., USSR). *Klin. Khir.* (4):35-38, 1975.

6417 CALCULOSIS ON SILK LIGATURE IN THE COMMON BILE DUCT. *(Rus.)* Prikupets, V. L.; Sizemov, O. D. (Moscow Med. Stomatol. Inst., USSR). *Khirurgiia* (1):138-139, 1975.

6418 BIOELECTRIC ACTIVITY OF THE STOMACH AND SMALL INTESTINE AFTER CHOLECYSTECTOMY. *(Rus.)* Oleksin, V. I.; Kuziv, K. F. (Rozhniatovsk Centr. Reg. Hosp., Ivano-Frankovsk, USSR). *Klin. Khir.* (1):64-66, 1975.

6419 NONPARASITIC HEPATIC CYST IN COMBINATION WITH MALIGNANT ADENOCYSTOMAS IN THE COMMON BILE DUCT. *(Rus.)* Abakumov, M. M.; Davidovskaia, N. I.; Pogodina, A. N. (N. V. Sklifosovskii Res. Inst. First Aid, Moscow, USSR). *Khirurgiia* (1):137-138, 1975.

6420 THE TREATMENT OF GALLBLADDER AND BILIARY DISEASES WITH COLRHEO^{R} (THEOPHYLLINE SODIUM GLYCINATE--1.074 g; MESO-INOSITOL--2.700 g; MAGNESIUM SULFATE--18.900 g). *(Ger.)* Bornemann, W.; Szonn, D. (no affil.). *Z. Ther.* 12(6):366-369, 1974.

6421 FEATURES OF BLOOD SERUM IN OBSTRUCTIVE JAUNDICE. *(Rus.)* Chernova, G. V.; Tokarskaia, Z. V. (Inst. Biophysics, Moscow, USSR). *Lab. Delo* (3):140-142, 1975.

6422 PROGNOSIS FOR CHOLELITHIASIS IN THE AGED. *(Fr.)* Fouet, P.; Denez, B.; Kokotek, H.; Zimmermann, P. (Albert Chenevier Hosp., Creteil, France). *Ann. Med. Interne (Paris)* 126(3):193-195, 1975.

6423 RARE CASE OF A CARCINOSARCOMA OF THE GALLBLADDER. *(Ger.)* Stempinski, E.; Beger, H. C. (Westend Clin., Free Univ., Berlin, Germany). *Z. Gastroenterol.* 12(6):485-489, 1974.

6424 DIAGNOSIS OF BACTERIAL INFECTIONS IN THE BILIARY TRACT AND SPECIFIC CHEMOTHERAPY. *(Ger.)* Tauchnitz, C.; Horn, M.; Storch, W. (Med. Clin., Karl Marx Univ., Leipzig, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):129-133, 1974.

6425 COMPLICATIONS IN BILIARY SURGERY AND REPEATED BILIARY SURGERY. *(Ger.)* Zangl, A. (Provincial Hosp., Salzburg, Austria). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):135-140, 1974.

6426 FUNCTIONAL DISORDERS OF THE BILIARY TRACT. *(Ger.)* Ritter, U. (Med. Coll., Lubeck, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4): 119-123, 1974.

6427 DISEASES OF THE INTRAHEPATIC AND EXTRAHEPATIC BILIARY TRACT. *(Ger.)* Emmrich, R. (Med. Clin., Karl Marx Univ., Leipzig, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):85-92, 1974.

6428 BILIARY DISEASE AND ITS EFFECT ON THE LIVER. *(Ger.)* Petzold, H. (Med. Clin., Karl Marx Univ., Leipzig, Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):93-97, 1974.

6429 JAUNDICE IN BILIARY COLIC. *(Ger.)* Fodor, O.; Dancea, S.; Goia, A.; Ban, A. (Med. Clin., Cluj, Rumania). *Dtsch. Z. Verdau. Stoffwechselkr.* 34(2-4):105-107, 1974.

6430 A SURVEY OF 256 CASES OF SO-CALLED ADENOMYOMATOSIS OF THE FUNDUS OF THE GALLBLADDER. *(Fr.)* Arianoff, A. A.; Vielle, G.; Delacroix, G.; Dewulf, E.; Arianoff, V. (Saint Elisabeth Clin., Brussels, Belgium). *Acta Gastroenterol. Belg.* 37(9):429-447, 1974.

6431 CONGENITAL ABSENCE OF THE GALLBLADDER. *(Pol.)* Zwierz, J.; Biernacky, J.; Malinowski, J. (Munic. Hosp., Zgierz, Poland). *Wiad. Lek.* 28(5):407-408, 1975.

6432 CARCINOMAS OF THE EXTRA-HEPATIC BILE DUCTS. *(Fr.)* Arianoff, A. A.; Vielle, G.; Dewulf, E.; Arianoff, V. (Sainte Elisabeth Clin., Brussels, Belgium). *Acta Chir. Belg.* 74(1):45-62, 1975.

6433 CHOLELITHOLYSIS: PRINCIPLES AND RESULTS. *(Ger.)* Paumgartner, G. (Inst. Clin. Pharmacol., Univ. Bern, Switzerland). *Z. Gastroenterol.* 13(suppl.):295-299, 1975.

6434 CONTROLLING HEMORRHAGE AND BILE DISCHARGE AFTER CHOLANGIOGRAPHY PERFORMED BY PERCUTANEOUS PUNCTURE OF THE LIVER. *(Fr.)* Krajka, C.; Dybicki, G. (Acad. Med., Gdansk, Poland). *Lyon Chir.* 71(2):130-132, 1975.

6435 ANASTOMOSIS OF THE INTRAHEPATIC BILE DUCT WITH THE DIGESTIVE TRACT IN OBSTRUCTION OF THE COMMON BILE DUCT. *(Bul.)* Kondova, P.; Mateev, V. (N. I. Pirogov Emergency Med. Inst., Sofia, Bulgaria). *Khirurgiia* 27(3):245-247, 1974.

6436 ON THE DIAGNOSIS OF HEMOBILIA. *(Rus.)* Karaman, N. V.; Kravchuk, A. N.; Zimovsky, V. L. (N. I. Pirogov Odessa Med. Inst., USSR). *Vestn. Khir.* 114(3):40-42, 1975.

6437 IN DEFENSE OF CHOLECYSTOSTOMY. *(Rus.)* Zhitnjuk, R. I. (Dept. General Surg., S. M. Kirov Military Med. Acad., Leningrad, USSR). *Vestn. Khir.* 114(3):36-40, 1975.

6438 RARE FISTULOUS COMPLICATIONS OF CALCULOUS CHOLECYSTITIS WITH THE CONTRIBUTION OF THREE CASES. *(Bul.)* Dobrev, J.; Mltadenov, V. (Fac. Med., Sofia, Bulgaria). *Chirurgia* 27(5):372-379, 1974.

6439 CHOLELITHIASIS IN CHILDREN. *(Pol.)* Wojtowicz, M.; Bartkowiak, Z.; Jankowski, A. (Clin. Pediatr. Surg., Inst. Ped. Med. Acad., Pozuani, Poland). *Pol. Przegl. Chir.* 46(12a):213-216, 1974.

6440 THE MORPHOLOGY OF GALLSTONES. *(Ger.)* Wolpers, C. (Inst. Med. Phys., Univ. Munster, Germany). *Z. Gastroenterol.* 13(suppl):302-303, 1975.

6441 INDICATIONS FOR AND RESULTS FROM SURGERY AND REPEATED SURGERY ON THE GALLBLADDER AND BILIARY TRACT. *(Ger.)* Gruenagel, H. H.; Krause, W. D. (Lutheran Hosp., Dusseldorf, Germany). *Med. Welt* 25(35):1377-1383, 1974.

6442 PROBLEMS IN THE DIAGNOSIS AND TREATMENT OF SPONTANEOUS CHOLECYSTOGASTRIC FISTULAS. *(Ger.)* Dinstl, K.; Thetter, O. (1st Surg. Clin., Univ. Vienna, Austria). *Zentralbl. Chir.* 99(31):967-971, 1974.

6443 ANTIBIOTIC TREATMENT OF CHOLECYSTITIS AND CHOLANGITIS VIA THE UMBILICAL VEIN. *(Hun.)* Farkas, A.; Molnar, J.; Tolnai, K. (Dept. Surg., Budapest XXI Dist. Munic. Hosp., Hungary). *Magy. Sebesz.* 27(6):374-376, 1974.

6444 THE TECHNIQUE OF REDUCING THE MORTALITY RATE IN SURGERY ON EXTRAHEPATIC BILE DUCTS. *(Rus.)* Kurbangaleev, S. M.; Rall, M. B. (1st Leningrad Med. Inst., USSR). *Vestn. Khir.* 114(2):37-42, 1975.

6445 RESECTION OF EXTRAHEPATIC BILE DUCTS IN CANCER PATIENTS. *(Rus.)* Futorian, E. S.; Shubin, B. M. (P. A. Gertsen Inst. Oncol., Moscow, USSR). *Vestn. Khir.* 114(1):37-41, 1975.

6446 ROENTGENOSURGICAL CORRELATIONS IN CHOLEDOCHOLITHIASIS. *(Rus.)* Ginzburg, S. N.; Dzhioev, V. G. (Moscow Med. Stomatol. Inst., USSR). *Vestn. Khir.* 114(1):41-44, 1975.

6447 DUODENAL TONUS IN CERTAIN DISEASES OF THE GALLBLADDER. *(Rus.)* Savinov, I. P.; Danilov, L. N. (Sci. Res. Inst. Pulmonology, Leningrad, USSR). *Vestn. Khir.* 114(1):44-46, 1975.

6448 BENIGN DIFFUSE FIBROCHOLANGIOMA. BILIARY MICROHAMARTOMA. *(Fr.)* Caroli, J.; Hadchouel, P.; Huguet, C. (St. Antoine Hosp., Paris, France). *Med. Chir. Dig.* 3(6):367-369, 1974.

6449 THE SURGICAL PROBLEM OF CHOLEDOCHODUODENOSTOMY. *(Ger.)* Grill, W. (no affil.). *Fortschr. Med.* 93(6):251-253, 1975.

6450 INDICATIONS FOR CHOLEDOCHODUODENOSTOMY OVER SPHINCTEROTOMY. *(Ger.)* Hess, W. (no affil.). *Fortschr. Med.* 93(6):250, 1975.

6451 MULTIPLE OPERATIONS ON THE BILE DUCTS. *(Ger.)* Grill, W. (no affil.). *Fortschr. Med.* 93(6):248-250, 1975.

6452 GALLBLADDER PERFORATION SEEN AS A RENAL TUMOR. *(Ger.)* Haas, H.; Senge, T.; Schulte, G. A. (Recklingshausen Hosp., Germany). *Z. Urol. Nephrol.* 67(12):937-940, 1974.

6453 UNILOBULAR CYSTIC DILATATION OF A BILE DUCT SEGMENT (CAROLI'S DISEASE) WITH MICROSCOPIC CALCULI. *(Fr.)* Caroli, J.; Laverdant, C.; Moine, D.; Hepp, J.; Hadchouel, P. (St. Antoine Hosp., Paris, France). *Med. Chir. Dig.* 3(6):371-375, 1974.

6454 THE CONTENT OF BILE ACIDS IN THE BILE OF PATIENTS WITH CHRONIC CHOLECYSTITIS. *(Rus.)* Muradkasymov, I. R.; Bakhadyrov, K. B. (Tashkent Med. Inst., USSR). *Klin. Med.* 53(1):80-83, 1975.

6455 INDICATIONS FOR CHOLEDOCHODUODENOSTOMY OVER PAPILLOTOMY: COMPLICATIONS AND MORTALITY. *(Ger.)* Fodisch, H. J. (no affil.). *Fortschr. Med.* 93(6):250-251, 1975.

6456 MEDICAL TREATMENT OF BILIARY CALCULI WITH CHENODEOXYCHOLIC ACID. PRELIMINARY EXPERIENCE WITH DIFFERENT MEANS OF ADMINISTRATION. *(It.)* Garagnani, A.; Evangelisti, G. B.; Faggioli, M.; Mazzetti, M. (S. Maria della Scaletta, Imola, Italy). *Clin. Ter.* 72(5):435-444, 1975.

6457 MINERAL WATER IN THE THERAPY OF BILIARY DISEASES. *(Ger.)* Boecker, W. (Sanit. Frauenburg, Bad Mergentheim, Germany). *Therapiewoche* 25(9):1048-1054, 1975.

6458 COMPLICATIONS FOLLOWING SURGICAL INTERVENTION ON THE GALLBLADDER AND BILIARY TRACT. *(Ger.)* Markoff, N.; Hobl, E. (Canton. Hosp., Chur, Switzerland). *Therapiewoche* 25(9):1057-1070, 1975.

6459 SPONTANEOUS BILIARY FISTULA. *(It.)* Bergamaschi, R.; Dallari, A.; Coppi, G.; Esposito, A.; Melotti, G. (Hosp. Inst. Modena, Italy). *Terapia* 60(431):22-31, 1975.

6460 BILIARY DISEASE IN PRACTICE--DIAGNOSTIC PROBLEMS AND THERAPEUTIC CONSEQUENCES. *(Ger.)* Kremp, F. (Munic. Hosp., Karlsruhe, Germany). *Therapiewoche* 25(9):1016-1021, 1975.

6461 DIAGNOSIS AND CONSERVATIVE THERAPY OF BILIARY TRACT DISEASES. *(Ger.)* Schirmeister, J. (Munic. Hosp., Karlsruhe, Germany). *Therapiewoche* 25(9):1022-1028, 1975.

6462 PATHOLOGICAL ANATOMY OF BILIARY TRACT DISEASES AND THEIR COMPLICATIONS. *(Ger.)* Bassler, R.; Gronemeyer, R. (Munic. Hosp., Fulda, Germany). *Therapiewoche* 25(9):1000-1015, 1975.

6463 DIAGNOSIS AND TREATMENT OF COMMON BILE DUCT STONES. *(It.)* Tagliacozzo, S. (Surg. Clin., Univ. Cagliari, Italy). *Rass. Med. Sarda* 76:309-312, 1974.

6464 GRANULAR CELL MYOBLASTOMA OF THE CYSTIC DUCT. A CASE ASSOCIATED WITH HYDROPS OF THE GALLBLADDER. *(E.)* Reul, Jr., G. J.; Rubio, P. A.; Berkman, N. L. (Baylor Coll. Med., Houston, Tex.). *Am. J. Surg.* 129(5):583-584, 1975.

6465 BACTERIOLOGY OF THE GALLBLADDER BILE IN NORMAL SUBJECTS. *(E.)* Csendes, A.; Fernandez, M.; Uribe, P. (Hosp. Jose Joaquin Aguirre, Santiago, Chile). *Am. J. Surg.* 129(6):629-631, 1975.

6466 CARCINOMA OF BILIARY TREE: III. CARCINOMA OF AMPULLA OF VATER. *(E.)* Thorbjarnarson, B. (New York Hosp., Cornell Med. Cent., Ithaca). *N.Y. State J. Med.* 75(6):860-862, 1975.

6467 THE EFFECT OF METOCLOPRAMIDE ON THE CONTRACTION OF THE HUMAN GALLBLADDER. *(E.)* Katevuo, K.; Kanto, J.; Pihlajamaki, K. (Dep. Radiol., Turku Univ., Finland). *Invest. Radiol.* 10(3):197-199, 1975.

6468 MANAGEMENT OF GALLSTONES IN THE AGED. *(E.)* Ratner, J. T.; Rosenberg, G. M. (Maimonides Hosp., Montreal, Canada). *J. Am. Geriatr. Soc.* 23(6):258-264, 1975.

6469 ODDI SPHINCTEROPLASTY IN THE MANAGEMENT OF COMPLICATED BILIARY AND PANCREATIC DISEASE. *(E.)* Haff, R. C.; Torma, M. J. (Wilford Hall USAF Med. Cent., Lackland Air Force Base, Tex.). *Am. J. Surg.* 129(5):509-512, 1975.

6470 DILATATION OF BILIARY TRACT STRICTURES. A NEW ROENTGENOLOGIC TECHNIQUE. *(E.)* Burhenne, H. J. (Child. Hosp., San Francisco, Calif.). *Radiol. Clin.* 44(3):153-159, 1975.

6471 CHOLECYSTECTOMY IN ACUTE AND CHRONIC CHOLECYSTITIS: A FIVE-YEAR STUDY. *(E.)* Viti, G. (no affil.). *Abdom. Surg.* 17(5):130-132, 1975.

6472 TECHNIQUES OF OPERATIVE CHOLANGIOGRAPHY. *(E.)* Tinckler, L. F. (Maclor Gen. Hosp., Wrexham, England). *J. R. Coll. Surg. Edinb.* 19(5):316-322, 1974.

6473 ACUTE ACALCULOUS CHOLECYSTITIS: COMPLICATION OF OTHER ILLNESSES IN CHILDHOOD. *(E.)* Ternberg, J. L.; Keating, J. P. (Washington Univ. Sch. Med., St. Louis, Mo.). *Arch. Surg.* 110(5):543-547, 1975.

6474 A CONTROLLED TRIAL OF PARENTERAL PROPHYLACTIC GENTAMICIN THERAPY IN BILIARY SURGERY. *(E.)* Keighley, M. R. B.; Baddeley, R. M.; Burdon, D. W.; Edwards, J. A. C.; Quoraishi, A. H.; Oates, G. D.; Watts, G. T.; Alexander-Williams, J. (Gen. Hosp., Birmingham, England). *Br. J. Surg.* 62(4):275-279, 1975.

6475 SPONTANEOUS RETROPERITONEAL PERFORATION OF THE GALLBLADDER. *(E.)* Horovitz, I. L. (Rothschild Univ. Hosp., Haifa, Israel). *Int. Surg.* 60(4):235-236, 1975.

See also, 5370, 5380, 5517, 5624, 5631, 5640, 5643, 5646, 5653, 5662, 5674, 5676, 5678, 5701, 5820, 5864, 5873, 5891, 5967, 6034, 6132, 6190, 6323, 6326, 6557.

6476 UNUSUAL FEATURES AND COMPLICATIONS OF BEZOARS IN CHILDREN. *(E.)* Harris, V. J.; Hanley, G. (Cook Cty. Child. Hosp., Chicago, Ill.). *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 123(4): 742-745, 1975.

Detailed case histories are presented for three children with trichobezoars, and four others with bezoars are reported. The children (six girls and one boy) ranged in age from 15 months to 9 yr. A 3 x 3 cm mass was palpable in the left hypochondrium of the abdomen of the first case, a 4-yr-old girl, and another, 1.5 x 2 cm, was palpable in the left umbilical area. Both were smooth, nontender, superficial and mobile. A plain film demonstrated a radiopaque mass within the stomach, and segments were also seen in the jejunum, suggesting intussusception. The specimen (22 x 5 cm) was removed at surgery. Plain roentgenograms of the abdomen demonstrated a mass in the stomach in the second patient (a 15-month-old girl), as well as multiple small bowel fluid areas with an overall appearance suggestive of small bowel obstruction. At surgery a large comma-shaped trichobezoar (80 g) was removed from the stomach and multiple intussusceptions were reduced. An upper gastrointestinal series in the third case (a 6-yr-old girl) showed a large gastric bezoar, and perforations in the posterolateral and superomedial portions of the fundus of the stomach. At surgery a 300 g, foul-smelling trichobezoar was removed from the stomach. In addition, two perforations of the anterior wall of the stomach were found, and a large pelvic abscess was drained. The postoperative course was complicated by pneumonia, and the patient died. All seven patients seen had vomiting and abdominal pain as the most common presenting symptom, although anorexia, nausea, weakness, weight loss and constipation were also commonly seen. One patient unexpectedly passed a bezoar *via* the rectum after a 1-yr history of vomiting. Two of the children were mentally retarded, and the others were in need of psychiatric help.

6477 ASSESSMENT OF SIMPLE METHODS OF MEASURING INTESTINAL TRANSIT TIMES IN CHILDREN WITH GASTROENTERITIS. *(E.)* Higgs, R. H.; Ellis-Pegler, R. B.; Lambert, H. P. (St. George's Hosp., London, England). *Gut* 16(6):458-461, 1975.

Intestinal transit times were measured in 11 children (aged 3-21 months) with gastroenteritis using carmine suspension and radioopaque pellets. The 2 methods gave widely divergent results. Sick children, often dehydrated and passing frequent fluid motions, were found by the carmine method to have rapid transit times consistent with the clinical picture (mean 2.5 hr). In contrast, the first pellets were seen over a range of 2-49 hr (mean 16.8 hr), similar to that found in controls without diarrhea. Other methods of measuring pellet transit times confirmed this anomaly. Clearly, the pellet method is unsuitable for use in gastroenteritis. It is suggested that the findings may be explained by sequestration of pellets in a particular area of the bowel such as stomach or cecum, or as an example of streaming of gut contents into solid and liquid phases. Pellets might thus be expected to travel in the slower solid phase. Carmine, as a solid suspension, is usually regarded as a solid phase marker, but the results of this study suggest that in acute gastroenteritis this substance travels with the liquid phase of the gut contents. No clear relation was noted between transit time, percentage dehydration, or the presence of reducing substance in the stool.

6478 SUCROSE IN ORAL THERAPY FOR CHOLERA AND RELATED DIARRHOEAS. *(E.)* Nalin, D. R. (Cholera Res. Lab., Dacca, Bangladesh). *Lancet* 1(7922):1400-1402, 1975.

Sucrose was tested as a possible alternative to glucose in p.o. diarrhea therapy in 18 patients (17 men and 1 woman, aged 11-65 yr), including 13 with cholera. Each patient received p.o. sucrose plus electrolytes as a maintenance solution. All received tetracycline (250 mg, p.o., every 6 hr for 4 days) from the start of p.o. maintenance. Fifteen of the patients could be maintained using this solution, but 3 developed massive increases in net fluid losses and increases in plasma specific gravity, necessitating termination of p.o. therapy. It is suggested that these 3 patients may not have been able to hydrolyze sufficient sucrose to yield the amount of glucose needed for adequate absorption; this could account for the presence of sugar in the stool and could be attributed to the type of disaccharidase deficiency reported to occur during and after diarrheal episodes. Of 12 patients tested, all had significant concentrations of stool reducing sugar. These results using sucrose are in striking contrast to results with p.o. glucose-electrolyte solution. However, it is suggested that sucrose-electrolyte solution could be used successfully to treat most patients under emergency conditions, in which i.v. fluid supplies have to be conserved and glucose is not available.

6479 ACETYLSALICYLIC ACID AND GASTROINTESTINAL BLEEDING MEASUREMENT OF BLOOD LOSS USING A MODIFIED RADIOACTIVE CHROMIUM METHOD. *(E.)* Arvidsson, B.; Magnusson, B.; Solvell, L.; Magnusson, A. (Radiat. Phys. Dep., Univ. Goteborg, Sweden). *Scand. J. Gastroenterol.* 10:115-160, 1975.

The fecal blood loss caused by a new acetylsalicylic acid preparation containing buffering substances was compared with the bleeding induced by plain acetylsalicylic acid tablets. Blood loss was quantified using a ^{51}Cr-labeling technique. Eighteen healthy male volunteers (aged 26-56 yr) were given one of two different salicylate preparations administered crossover in randomized order during two 7-day periods. One drug was a plain acetylsalicylic acid tablet containing 0.5 g acetylsalicylic acid. The other was a buffered tablet containing 0.5 g acetylsalicylic acid, 1.25 g sodium bicarbonate, and 0.5 g citric acid. The dosage was 1 g acetylsalicylic acid, 3 times/day from day 1-7. Two tablets were taken immediately before breakfast, two immediately before lunch, and two at 1 hr before dinner. The plain acetylsalicylate acid was swallowed whole with half

a glass of water. The buffered preparation was first dissolved in the same amount of water. An interval of 3 weeks was allowed between the two different drugs. While receiving plain acetylsalicylic acid, 5 out of 18 subjects bled more than 10 ml daily. Three bled more than 20 ml daily. When receiving buffered acetylsalicylic acid, no subject bled 20 ml or more, and only 3 bled between 10 and 20 ml daily. Thus, a median daily blood loss of 4.6 ml during intake of plain acetylsalicylic acid was decreased to 2.0 ml during intake of the buffered preparation. The findings of this study confirm previous findings that increasing buffering capacity will successively decrease the amount of gastrointestinal blood loss. In this present study highly sensitive measuring equipment was used which made it possible to reduce the amount of ^{51}Cr from 1/3 to 1/10 of the previously used amounts.

6480 SERUM IgE IN EOSINOPHILIC GASTROENTERITIS. RESPONSE TO INTESTINAL CHALLENGE IN TWO CASES. *(E.)* Caldwell, J. H.; Tennenbaum, J. I.; Bronstein, H. A. (Ohio State Univ. Coll. Med., Columbus). *New Engl. J. Med.* 292(26):1388-1390, 1975.

Since serum levels of IgE have found wide application in the *in vitro* assessment of more common forms of allergy, e.g., ragweed sensitivity, a study of serum IgE was carried out in 2 patients with proved eosinophilic gastroenteritis. It was hoped to determine whether serum IgE in these patients correlated with historical evidence of food sensitivity and with results of intradermal testing. A 40-yr-old male had noticed gastrointestinal difficulty occurring within min after the ingestion of specific foods. His history revealed seasonal allergic rhinitis. There had been no correlation between upper-airway and digestive symptoms, and no relief of the latter by antihistamines which he took occasionally. Intradermal skin tests showed 2+ or greater reactions to grasses, ragweed, corn, oats, peas, barley, beef, egg, chicken, lamb, pork, and fish. Small bowel biopsy disclosed eosinophilia of the lamina propria and was otherwise normal. Since discharge he had been successfully treated with small doses of p.o. corticosteroids. Three yr later an assay of a serum sample for specific IgE antibodies by the radioallergosorbent test showed a 1+ score for fish, 2+ for ragweed, grass and corn, and 4+ for egg and wheat. In a second case, a 66-yr-old male was hospitalized because of eosinophilia and several weeks of postprandial diarrhea and abdominal distension. Gastroduodenal endoscopy was carried out, and biopsy showed dense infiltration of the pyloric and proximal duodenal mucosa with eosinophils and an otherwise normal mucosa. Intradermal tests for numerous inhalants and food allergies were negative. Skin tests for mumps and histoplasmin were positive at 48 hr. He was treated with low doses of oral corticosteroids with improvement for a period of 2 yr. Intestinal challenge studies caused the serum IgE levels in the former patient, already markedly elevated in the fasting state, to rise nearly 3-fold within the hr after challenge with food to which he was clinically sensitive. Challenge of the latter patient evoked no symptoms and no change in the normal base-line level of IgE. These data indicate that a mechanism other than classic immediate hypersensitivity was involved in the second patient's illness. In contrast, the first patient appeared to have true IgE-mediated food allergy.

6481 EARLY RECOVERY OF EXOCRINE PANCREATIC FUNCTION IN ADULT PROTEIN-CALORIE MALNUTRITION. *(E.)* Kumar, R.; Banks, P. A.; George, P. K.; Tandon, B. N. (All India Inst. Med. Sci., New Delhi). *Gastroenterology* 68(6):1593-1595, 1975.

The early pattern of recovery of pancreatic exocrine function in response to a high protein diet was studied in 9 patients (6 men and 3 women, aged 20-70 yr) whose dietary histories indicated prolonged intake of food low in calories and deficient in protein. All patients showed symptoms of diarrhea, weakness, and peripheral edema. Pancreozymin-secretin tests were performed. The patients were placed on a diet which provided 50 cal/kg composed of 20% calories from animal protein of high biological value, 55% calories from fat, and 25% calories from carbohydrate. Repeat studies of pancreatic exocrine function were performed at 2 weeks and at 6 weeks after dietary therapy was instituted. The volume of basal and stimulated pancreatic flow was not significantly altered by the high-protein diet. There was significant improvement in the maximal bicarbonate concentration at 2 weeks but no additional improvement at 6 weeks. The maximal bicarbonate concentration of 71.3 mEq/liter at 2 weeks and 76.0 mEq/liter at 6 weeks are still subnormal compared with control volunteers in India. A significant improvement in protease secretion after 2 weeks was sustained but not increased after 6 weeks on the high-protein diet. The results of this study confirm the characteristic secretory deficit in adult protein caloric malnutrition and reveal that there is substantial improvement in pancreatic exocrine function after only 2 weeks of dietary therapy with improvement in maximum bicarbonate concentration and increased protease secretion. This rapid improvement indicates that there is considerable regenerative ability in the pancreas of man despite long-standing severe protein calorie malnutrition.

6482 DIFFERENTIAL MEASUREMENT OF SMALL AND LARGE BOWEL TRANSIT TIMES IN CONSTIPATION AND DIARRHOEA: A NEW APPROACH. *(E.)* Waller, S. L. (Cent. Middlesex Hosp., London, England). *Gut* 16(5): 372-378, 1975.

A method which allows the transit time through the small and large intestine to be differentiated was studied in 13 patients. Seven of the patients had diarrhea, which was caused by the irritable bowel syndrome (three), vagotomy and pyloroplasty (three), and laxative abuse (one). Just before breakfast each patient received a radiotelemetering capsule containing a disc of filter paper impregnated with 5 to 10 μCi of ^{51}Cr. After breakfast each was given 25 radioopaque pellets. The position of the capsule was plotted at intervals from zero to 10 and 12 hr daily. Plots were made every 15 to 20 min for the stomach and small intestine and every 30 to 60 min for the colon. Gastric emptying and small intestinal transit were similar in all patients. In con-

trast, colonic transit was faster in patients with diarrhea (17.5 hr) than in controls (118 hr). Senna was administered to four constipated subjects and produced a transit plot and elimination of the pellets and capsule which resembled diarrheal states in two of the four. In the other two patients, senna caused neither movement of the capsule nor bowel action. One diarrheal subject was restudied while taking codeine phosphate (60 mg over a 6-hr period). Small intestinal transit time was prolonged by only 2 hr, but the capsule remained in the colon an extra 34 hr. It is concluded that this new method allows differential measurement of small and large bowel transit times in man, thus allowing frequent observations of movements of colonic contents to be made without recourse to radiographs. The method is time consuming, but technically simple, avoids intubation, and gives much information with minimal irradiation of the patient. It is concluded that fast colonic transit is a striking feature of diarrhea.

6483 RELATIVE ABUNDANCE OF BIG AND LITTLE GASTRINS IN THE TUMOURS AND BLOOD OF PATIENTS WITH THE ZOLLINGER-ELLISON SYNDROME. *(E.)* Dockray, G. J.; Walsh, J. H.; Passaro, E., Jr. (VA Wadsworth Hosp. Cent., Los Angeles, Calif.). *Gut* 16(5):353-358, 1975.

The relative concentrations of big gastrin (G-34) and little gastrin (G-17) were compared in the sera and tumors (gastrinomas) of five Zollinger-Ellison syndrome patients. Big and little gastrins were identified in all ten serum samples and in all ten tumor biopsies examined. In serum, G-34 (range of concentrations, 58 to 220,000 fM/ml) was the major form of gastrin and G-17 (22 to 78,000 fM/ml) was a minor component. The mean relative abundance of G-17 (G17/[G17 + G34]) in serum was 0.18 and the mean relative abundance of G-34 was 0.82. In tumor, however, the opposite was true. G-17 (49 to 869,000 pM/g) was the major component and G-34 (45-464 pM/g) a minor component, and the relative proportions of G-17 and G-34 were 0.73 and 0.27, resp. Following an i.v. injection of porcine secretin (2.0 U/kg), there was a rapid increase in concentration of all forms of gastrin in the blood, but the increase in G-17 was proportionately greater than that of G-34 (relative abundance of G-17 in basal serum was 0.21 compared with 0.37, 5 min after secretin administration). Differences in the half lives of G-17 and G-34 may partly explain their relative abundancy in serum and tumor tissues. It is noted that G-17 is about seven times more potent than G-34 in stimulating acid secretion when potencies are expressed in terms of serum concentration. Thus, although G-17 constituted only about 20% of the serum gastrin immunoreactivity, its contribution to the biological activity in serum was the most important circulating form of gastrin.

6484 KAPOSI'S SARCOMA OF THE STOMACH AND DUODENUM: ENDOSCOPIC AND ROENTGENOLOGIC CORRELATIONS. *(E.)* Ahmed, N.; Nelson, R. S.; Goldstein, H. M.; Sinkovics, J. G. (M. D. Anderson Hosp. Tumor Inst., Houston, Tex.). *Gastrointest. Endosc.* 21(4):149-152, 1975.

To stress the occurrence, clinical presentation and management of gastrointestinal involvement with Kaposi's sarcoma, with particular emphasis on the endoscopic and radiological means of diagnosis and evaluation during treatment, four case reports are presented. Biopsies confirmed the diagnosis of Kaposi's sarcoma in the first case, a 68-yr-old man. Physical examination revealed widespread typical cutaneous lesions, hepatomegaly, diminished peripheral pulses in the lower extremity, and a right pleural effusion yielding Karposi's sarcoma cells on cytological examination. Gastroduodenoscopy showed numerous, purple macular lesions in the body of the stomach, in addition to sessile polypoid lesions (0.5-1.5 cm in diameter). Chemotherapy produced total regression of lesions with only two tiny white polypoid areas in the body of the stomach; the skin lesions remained unchanged after an initial, partial regression. The second case (a 60-yr-old man) showed cutaneous lesions and splenomegaly. Gastroscopy showed several 1 to 2 cm lesions at the junction of the body and the fundus of the stomach, in addition to similar 4 to 5 mm lesions present more distally along the greater curvature. Chemotherapy produced complete regression of the gastric lesions, but produced no response in skin involvement. A double-contrast view of the stomach in the third case (a 77-yr-old man) showed bulbous enlargement of two gastric rugae and a small nodular filling defect along the lesser curvature. An upper gastrointestinal radiograph demonstrated a target lesion of the upper body of the stomach in the fourth case (a 65-yr-old man), composed of a submucosal filling defect with a central collection of barium. Both these patients were treated with chemotherapy, and the outcome awaits further evaluation. The majority of upper gastrointestinal lesions in Kaposi's sarcoma were found in the stomach at endoscopy. There are three distinct types: maculopapular; nodular and polypous; and nodular with central umbilication. It is concluded that the visceral involvement in Kaposi's sarcoma is probably a manifestation of multicentricity rather than metastasis.

6485 EFFECT OF MICROORGANISMS ISOLATED FROM THE UPPER GUT OF MALNOURISHED CHILDREN ON INTESTINAL SUGAR ABSORPTION IN VIVO. *(E.)* Gracey, M.; Burke, V.; Thomas, J. A.; Stone, D. E. (Princess Margaret Children's Medical Res. Foundation, Perth, Western Australia). *Am. J. Clin. Nutr.* 28(8):841-845, 1975.

The effect of excessive numbers of microorganisms, cultured from the duodenum of malnourished children, on the intestinal transport of sugar was investigated. Adult Wistar rats (150-300 g) were used and a 20 cm segment of the jejunum was selected as a test site for the absorption of arbutin (p-hydroxyphenyl-β-glucoside). Several varieties of microorganisms were capable of interfering with absorption of sugar: lactobacillis, all species of *E. coli, Klebsiella, Pseudomonas, C. albicans* and *C. parapsilosis.* Cultures of *Staphylococcus saprophyticus, Salmonella paratyphi, Shigella, Proteus* and *C. tropicalis* did not have an inhibitory effect. Whether the observed effects were due to direct action of the organisms or to a toxin or other metabolic by-product requires further study. The fact that some of the organisms connected with inhibition were not considered entero-

pathogenic suggests that concepts of enteropathogenicity may require revision.

6486 ENTEROTOXIGENIC Escherichia coli-ASSOCAITED DIARRHEAL DISEASE IN APACHE CHILDREN. *(E.)* Sack, R. B.; Hirschhorn, N.; Brownlee, I.; Cash, R. A.; Woodward, W. E.; Sack, D. A. (Univ. Oregon Med. Sch., Portland). *N. Engl. J. Med.* 292(20):1041-1045, 1975.

A search for intestinal enterotoxigenic *Escherichia coli* was made in 59 Apache children hospitalized with 64 episodes of acute diarrhea. *E. coli* isolated from acute-phase and convalescent-phase specimens of small bowel fluid and stool were tested in three recognized models: the adult rabbit ileal loop, infant rabbit, and the adrenal-cell assay. Enterotoxigenic strains were isolated from ten children during acute diarrheal episodes (16%). None were isolated from convalescent-phase specimens. None of 64 enteropathogenic serotypes of *E. coli* from 43 children with diarrhea, however, caused fluid production in the ileal-loop model. These results suggest that enterotoxigenic *E. coli* may be the cause of considerable diarrhea in this population and that the term "enteropathogenic" as applied to serotypes of *E. coli* needs to be redefined.

6487 CARCINOIDS AND CANCER SUSCEPTIBILITY. *(Fr.)* Giroux, L.; Laurencelle, L.; Lesage, R.; Delorme, F. (Hosp. Notre Dame, Montreal, Canada). *Union Med. Can.* 104(4):596-600, 1975.

6488 PRIMARY GASTRODUODENITIS IN CHILDREN. *(Rus.)* Mazurin, A. V.; Miroshnichenko, V. A.; Barchunova, N. N.; Tsvetkova, L. N.; Galaeva, S. S. (Children's Hosp. No. 3, N. I. Pirogov Second Moscow Med. Inst., USSR). *Vopr. Okhr. Materin. Det.* 20(3):23-28, 1975.

6489 ON THE DIAGNOSTIC SIGNIFICANCE OF THE STUDY INTO THE AUTOREGULATIVE MECHANISM RESPONSIBLE FOR INHIBITION OF GASTRIC SECRETION. *(Rus.)* Saakyan, A. G.; Makarova, L. A. (Pyatigorsk Sci. Res. Inst. Health Resorts Physiother., USSR). *Sov. Med.* (2):44-49, 1975.

6490 THE USE OF COLABON (H-4419) IN THE TREATMENT OF INFANTILE DIARRHEA. *(Por.)* Schmidt, B. J.; Bourgeois, B.; Fleury, J.; Filho, O.; Nascimento, J. S.; Nakamura, K. T.; Cardoso, A.; Pereira, J. L.; Gizaai, G. F.; Costa, C. D. (Clin. Pediatr., Fac. Med. Sorocaba, Sao Paulo, Brazil). *Pediatr. Prat.* 45(1-2):11-16, 1974.

6491 DIARRHEAS DUE TO INTOLERANCE TO LACTOSE. PRELIMINARY REPORT ON THE USE OF LACTOSE-FREE MILK. *(Por.)* Alves, N. (Fac. Med., Minas Gerais, Brazil). *Pediatr. Prat.* 45(1-2):1-8, 1974.

6492 ACUTE HEMORRHAGE OF THE UPPER GASTROINTESTINAL TRACT WITH REGARD TO DRUG-INDUCED LESION OF THE GASTRIC MUCOSA. *(Ger.)* Schwenke, E.; Schwenke, G.; Willgeroth, C. (Med. Clin., Magdeburg-Altstads Dist. Hosp., Germany). *Z. Gesamte Inn. Med.* 30(5):198-205, 1975.

6493 R 18553 IN THE TREATMENT OF ACUTE DIARRHEA IN THE YOUNG CHILD. *(Fr.)* Dubru, J. M.; Lambrechts, A. (Pediatr. Clin., Univ. Liege, Belgium). *Rev. Med. Liege* 29(19):579-581, 1974.

6494 SURGERY FOR MALIGNANT ULCEROGENIC TUMORS IN THE PANCREAS. *(Rus.)* Samokhvalov, V. I.; Kalashnikov, S. A. (S. M. Kirov Mil. Med. Acad., USSR). *Vestn. Khir.* 114(2):35-37, 1975.

6495 OBSERVATIONS ON NUCLEAR INCLUSIONS IN MIXED TUMORS OF THE PAROTID GLAND. *(It.)* Motta, G.; Riva, A. (Clin. Otorthinolaryngol, Univ. Cagliari, Italy). *Arch. Ital. Anat. Istol. Patol.* 45(1-3):71-101, 1974.

6496 A CASE OF CONGENITAL ABSENCE OF THE ABDOMINAL MUSCLES. *(Pol.)* Jakubowska, D. (Acad. Med., Warsaw, Poland). *Wiad. Lek.* 28(5):409-413, 1975.

6497 THE ROLE OF BILE ACIDS IN INTESTINAL DISEASE. *(Fr.)* Andre, J. (St. Antoine Hosp., Paris, France). *Med. Chir. Dig.* 3(6):427-430, 1974.

6498 IMMUNOLOGICAL ASPECTS OF GASTROINTESTINAL NEOPLASMS. *(Ger.)* Hess, M. W.; Zimmermann, A.; Brun Del Re, G.; Cottier, H. (Pathol. Inst., Univ. Bern, Switzerland). *Schweiz. Med. Wochenschr.* 105(18):570-575, 1975.

6499 INDICATIONS FOR RADIOTHERAPY FOR GASTROINTESTINAL TUMORS. *(Fr.)* Veraguth, P. (Radiol. Ther. Clin., Univ. Bern, Switzerland). *Schweiz. Med. Wochenschr.* 105(18):560-563, 1975.

6500 THE REACTION OF THE BONE MARROW TO SURGICAL INTERVENTION FOR GASTRODUODENAL BLEEDING. *(Rus.)* Potashov, L. V.; Sedov, V. M. (1st Leningrad Med. Inst., USSR). *Vestn. Khir.* 114(2):16-19, 1975.

6501 BLOOD CLOTTING AND HEMOSTATIC THERAPY OF ACUTE GASTROINTESTINAL BLEEDING. *(Rus.)* Zarivchatskii, M. F. (Perm Med. Inst., USSR). *Vestn. Khir.* 114(2):20-22, 1975.

6502 THE STUDY OF THE CARRIER STATE IN TYPHOID FEVER. *(Rus.)* Bilibin, A. F.; Khundanov, L. L. (2nd Moscow Med. Inst., USSR). *Vestn. Akad. Med. Nauk SSSR* (2):53-59, 1975.

6503 POSTOPERATIVE RENAL INSUFFICIENCY AFTER GASTROINTESTINAL SURGERY. *(Fr.)* Martin-Dupont, M. C. (no affil.). *Bordeaux Med.* 7(16): 2351-2356, 1974.

6504 THE PRESENT STATE OF ENTERIC INFECTIONS. *(Sp.)* de Araujo Moraes, N. L. (Fac. Med. Sci., State Univ. Guanabara, Brazil). *Bol. Of. Sanit. Panam.* 78(4):294-306, 1975.

6505 WHAT IS GUARANTEED IN THE THERAPY OF CHRONIC INFLAMMATORY DISEASE OF THE LARGE INTESTINE? *(Ger.)* Miller, B.; Martini, G. A. (Med. Clin. II, Univ. Dusseldorf, Germany). *Internist (Berlin)* 15(12):600-604, 1974.

6506 IMMUNOLOGY IN GASTROENTEROLOGY. *(Ger.)* Warnatz, H. (Immunol. Clin., Univ. Erlangen, Nurnberg, Germany). *Fortschr. Med.* 93(6):255-258, 260-261, 1975.

6507 RESULTS WITH SOYA MILK IN GASTROENTERIC DISEASES IN INFANTS. *(It.)* Fiore, M. R.; Caputo, M.; De Luca, V. (A. Cardarelli Hosp., Naples, Italy). *Minerva Pediatr.* 26(26):1313-1317, 1974.

6508 UPPER GASTROINTESTINAL BLEEDING: CAUSES, DIAGNOSIS AND THERAPY. *(Ger.)* Phillip, J. (Med. Clin. Polyclin., Univ. Erlangen-Nuremberg, Erlangen, Germany). *Fortschr. Med.* 93(7):309-312, 1975.

6509 PATHOPHYSIOLOGY OF DIARRHEA. *(Ger.)* Ewe, K. (Med. Clin., Univ. Mainz, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:309-319, 1974.

6510 COMPARATIVE STUDIES OF ORAL AND SUBCUTANEOUS IMMUNIZATION AGAINST TYPHOID AND PARATYPHOID FEVER. *(Rus.)* Krasnoproshina, L. I. (I. I. Mechnikov Moscow Inst. Vaccines Sera, USSR). *Acta Microbiol. Acad. Sci. Hung.* 21(1-2):151-160, 1974.

6511 ANIMAL AND HUMAN STUDIES OF ORAL VACCINATION AGAINST TYPHOID AND PARATYPHOID FEVER. *(Rus.)* Meshalova, A. N.; Kurlova, V. I.; Ianisker, G. Ia.; Lavrovskaia, V. M.; Blant, M. E.; Negina, Iu. P. (I. I. Mechnikov Moscow Res. Inst. Vaccines Sera, USSR). *Acta Microbiol. Acad. Sci. Hung.* 21(1-2):167-171, 1974.

6512 CLINICAL AND IMMUNOLOGICAL CORRELATIONS IN TYPHOID FEVER AND FORMATION OF BACTERIA-CARRIER STATE. *(Rus.)* Bunin, K. V.; Bunin, M. V. (I. M. Sechenov 1st Moscow Med. Inst., USSR). *Klin. Med. (Mosk.)* 53(2):3-10, 1975.

6513 DETERMINATION OF IMMUNOGLOBULIN FRACTIONS IN THE BLOOD SERUM OF PATIENTS AND CARRIERS OF EL TOR VIBRIOS. *(Rus.)* Pokrovskii, V. I.; Reshetniak, T. V.; Bolshakova, N. Ia; Stefani, D. V. (Cent. Inst. Epidemiol., Moscow, USSR). *Zh. Mikrobiol. Epidemiol. Immunobiol.* (1):14-16, 1975.

6514 DISORDERS OF GASTROINTESTINAL MOTOR AND SPHINCTER FUNCTION. *(E.)* Cohen, S. (Hosp. Univ. Pennsylvania, Philadelphia). *Trans. Stud. Coll. Phys. Phila.* 42(4):389-392, 1975.

6515 GASTRINOMAS: GASTRIN-PRODUCING TUMORS. *(E.)* Stremple, J. F. (Univ. Pittsburgh Sch. Med., Pa.). *Surg. Clin. North Am.* 55(2):303-323, 1975.

6516 SPONTANEOUS PERFORATION OF THE GASTROINTESTINAL TRACT IN PATIENTS WITH CANCER. *(E.)* Lundy, J.; Sherlock, P.; Kurtz, R.; Fortner, J. G.; Turnbull, A. D. (Mem. Sloan Kettering Cancer Cent., New York, N.Y.). *Am. J. Gastroenterol.* 63(6):447-456, 1975.

6517 CARBOHYDRATE INTOLERANCE IN INFANTS WITH ACUTE DIARRHOEA AND ITS COMPLICATIONS. *(E.)* Chandrasekaran, R.; Kumar, V.; Walia, B. N. S.; Moorthy, B. (Postgrad. Inst. Med. Educ. Res., Chandigarh, India). *Acta Paediatr. Scand.* 64(3):483-488, 1975.

6518 DETECTION OF SALMONELLAE IN FOODSTUFFS, FECES, AND WATER BY IMMUNOFLUORESCENCE. *(E.)* Cherry, W. B.; Thomason, B. M.; Gladden, J. B.; Holsing, N.; Murlin, A. M. (Cent. Dis. Control., UDSA, Atlanta, Ga.). *Ann. NY Acad. Sci.* 254:350-368, 1975.

6519 RESULTS OF RADICAL RESECTION FOR PERIAMPULLARY CANCER. *(E.)* Warren, K. W.; Choe, D. S.; Plaza, J.; Relihan, M. (Lahey Clin. Fdn., Boston, Mass.). *Ann. Surg.* 181(5):534-540, 1975.

6520 FRONTIERS IN INFLAMMATORY BOWEL DISEASE. *(E.)* Shorter, R. G.; Shephard, D. A. E. (Mayo Clin., Rochester, Minn.). *Am. J. Dig. Dis.* 20(6):540-571, 1975.

6521 VARIABILITY OF SERUM GASTRIN LEVELS IN ZOLLINGER-ELLISON SYNDROME: STUDIES WITH TWO ANTISERA TO GASTRIN. *(E.)* Ivey, K. J.; Hansky, J. (R. Prince Alfred Hosp., Sydney, Australia). *Am. J. Dig. Dis.* 20(6):513-517, 1975.

6522 BEHCET'S DISEASE AND THE ALIMENTARY TRACT. *(E.)* Parkin, J. V.; Wight, D. G. D. (Addenbrooke's Hosp., Cambridge, England). *Postgrad. Med. J.* 51(594):260-264, 1975.

6523 CONCURRENCE OF ULCERATIVE COLITIS AND CHRONIC ACTIVE HEPATITIS. CLINICAL COURSES AND RESULTS OF COLECTOMY. *(E.)* Olsson, R.; Hulton, L. (Dep. Med. II, Sahlgrenska Hosp., Gotherburg, Sweden). *Scand. J. Gastroenterol.* 10(3):331-335, 1975.

6624 FINE STRUCTURE OF THE JEJUNAL ABSORPTIVE CELLS IN THE HUMAN INFANTS WITH PERSISTENT DIARRHEA. *(E.)* Yamamoto, T.; Fujita, M. (Dep. Anat., Univ. Kyushu, Fukuoka, Japan). *Nihon Rynsho Denshi Kenoikyo Gakkai-Shi* 7(3-4):341, 1974.

6525 A CONTROLLED CLINICAL TRIAL OF CHLORAMPHENICOL IN CHLORAMPHENICOL-RESISTANT TYPHOID FEVER. *(E.)* Calderon, E.; Gilman, R. H.; Snyder, M. J.; Vazquez, V.; Barreto, D. G.; Legorrieta, J.; Rodriguez, R.; Martinez, F.; Hornick, R. B. Woodward, T. E. (Hosp. Del Nino, Mexico City, Mexico). *Rev. Lat. Am. Microbiol.* 16(3):131-136, 1974.

6526 DIET AND INTESTINAL ENZYME ADAPTATION: IMPLICATIONS FOR GASTROINTESTINAL DISORDERS. *(E.)* Rosensweig, N. S. (St. Luke's Hosp. Cent., New York, N.Y.). *Am. J. Clin. Nutr.* 28(6):648-655, 1975.

6527 HUMAN IMMUNIZATION WITH KILLED CHOLERA VACCINE. *(E.)* Denchev, V.; Jeleva, M.; Duncheva, S. (Cent. Infect. Parasit. Dis., Sofia, Bulgaria). *Acta Microbiol. Acad. Sci. Hung.* 21(1-2): 209-212, 1974.

6528 IMMUNIZATION OF INFANTS AGAINST Escherichia coli ENTERITIS. *(E.)* Rauss, K.; Ketyl, I.; Szendrei, L.; Vertenyi, A. (Univ. Med. Sch., Pecs, Hungary). *Acta Microbiol. Acad. Sci. Hung.* 21(1-2): 181-185, 1975.

6529 GENERAL IMMUNITY REACTIONS AFTER ORAL ADMINISTRATION OF INACTIVATED TYPHOID VACCINES IN MAN. *(E.)* Karolcek, J.; Draskovicova, M.; Georch, D. (Res. Inst. Epidemiol., Bratislava, Czechoslovakia). *Acta Microbiol. Acad. Sci. Hung.* 21(1-2):143-150, 1975.

6530 ORAL IMMUNIZATION AGAINST DYSENTERY. *(E.)* Ketyi, I.; Rauss, K.; Vertenyi, A. (Univ. Med. Sch., Pecs, Hungary). *Acta Microbiol. Acad. Sci. Hung.* 21(1-2):81-85, 1974.

6531 SUCCESSFUL FIELD TRIALS WITH ORAL KILLED Shigella sonnei VACCINE. *(E.)* Grahneis, H. (Inst. Hygiene Halle Dist., W. Germany). *Acta Microbiol. Acad. Sci. Hung.* 21(1-2):75-80, 1974.

6532 IMMUNITY AGAINST DYSENTERY AFTER ENTERAL IMMUNIZATION WITH LIVE VACCINE. *(E.)* Denchev, V.; Trifonova, A.; Jeleva, M.; Duncheva, S.; Bratanova, V.; Dimitrova, Z.; Kovachev, P. (Cent. Infect. Parasit. Dis., Sofia, Bulgaria). *Acta Microbiol. Acad. Sci. Hung.* 21(1-2);71-74, 1974.

6533 ORAL IMMUNIZATION AGAINST ENTERIC INFECTIONS USING INACTIVATED BACTERIA. A REVIEW. *(E.)* Mochmann, H.; Ocklitz, H. W. (Inst. Inf. Dis. Child., Berlin, E. Germany). *Acta Microbiol. Acad. Sci. Hung.* 21(1-2):3-10, 1974.

6534 DEATH FROM PSEUDOMEMBRANOUS ENTEROCOLITIS. *(E.)* Ledger, W. J.; Puttler, O. L. (Univ. California Med. Cent., Los Angeles). *Obstet. Gynecol.* 45(6):609-613, 1975.

6535 SYNCHRONOUS MALIGNANT MUCOEPIDERMOID TUMOR OF THE PAROTID GLAND AND WARTHIN'S TUMOR IN ADJACENT LYMPH NODE. *(E.)* Lumerman, H.; Freedman, P.; Caracciolo, P.; Remigio, P. S. (Catholic Med. Cent., Brooklyn, N.Y.). *Oral Surg.* 39(6):953-958, 1975.

6536 REOVIRUS-LIKE PARTICLES IN JEJUNAL MUCOSA OF A JAPANESE INFANT WITH ACUTE INFECTIOUS NON-BACTERIAL GASTROENTERITIS. *(E.)* Suzuki, H.; Konno, T. (Tohku Univ. Sch. Med., Sendai, Japan). *Tohoku J. Exp. Med.* 115(3):199-211, 1975.

6537 THE PATHOGENESIS OF SHIGELLA DIARRHEA. V. RELATIONSHIP OF SHIGA ENTEROTOXIN, NEUROTOXIN, AND CYTOTOXIN. *(E.)* Keusch, G. T.; Jacewicz, M. (Mt. Sinai Sch. Med., City Univ. New York, N.Y.). *J. Infect. Dis.* 131:S33-39, 1975.

6538 SALMONELLOSIS IN INFANTS. *(E.)* Rocha, E. (Univ. Hosp., Mustapha, Algiers, Algeria). *Environ. Child Health* 21(113):60-63, 1975.

6539 DIPHENOXYLATE HYDROCHLORIDE (LOMOTIL) IN THE MANAGEMENT OF CHOLERA. *(E.)* De, S.; Sircar, B. K.; De, S. P. (Infect. Dis. Hosp., Calcutta, India). *Indian J. Med. Res.* 63(3):469-474, 1975.

6540 A COMPARATIVE EVALUATION OF THE TREATMENT OF TYPHOID FEVERS WITH CO-TRIMOXAZOLE AND CHLORAMPHENICOL IN EGYPT. *(E.)* Hassan, A.; Hathout, S.; Safwat, Y.; Erian, M.; Abdel Wahab, M. F.; Sorensen, K.; Sippel, J.; Farid, A. (Abbassia Fever Hosp., Cairo, Egypt). *J. Trop. Med.* 78(3):50-53, 1975.

6541 SURGERY IN GASTROINTESTINAL SCLERODERMA. SUBTOTAL COLECTOMY. *(E.)* Wehmeyer, D. L.; Kiser, J. (Wesley Med. Cent., Wichita, Kansas). *J. Kansas Med.* 76(6):129-131, 1975.

6542 ANEURYSM OF THE GASTRODUODENAL ARTERY. *(E.)* Amongero, F.; Edlin, P.; Gottesman, L.; Friedman, E. M.; Posevitz, L. (no affil.). *Angiology* 26(5):424-428, 1975.

6543 THE LIVER AND CARBOHYDRATE METABOLISM IN PROTEIN CALORIE MALNUTRITION. *(E.)* Kassem, A. S.; Hafez, M.; Badr-El-Din, M. K.; Hassan, A. H. I. (Dep. Pediatr., Alexandria, Egypt). *Environ. Child Health* 21(1):3-6, 1975.

See also, 5216, 5593, 5680, 5688, 5685, 5882, 5892, 5904, 6051.

6544 TRYPTIC ACTIVITY IN THE DUODENAL ASPIRATE FOLLOWING A STANDARD TEST MEAL IN GIARDIASIS. *(E.)* Chawla, L. S.; Sehgal, A. K.; Broor, S. L.; Verma, R. S.; Chhuttani, P. N. (Postgraduate Inst. Medical Education Res., Chandigarh, India). *Scand. J. Gastroenterol.* 10(4):445-447, 1975.

Tryptic activity was studied in the duodenal aspirate of giardiasis patients after a standard test meal. The diagnosis of giardiasis was made in 26 patients (17 men and nine women, aged 14-60 yr) by repeated stool examinations. Duodenal juice was collected for two hr after a meal containing 45 g glucose, 18 g soya bean oil, and 15 g dried milk protein powder in 300 ml warm water. Tryptic activity was estimated using synthetic α-N-benzoyl-L-arginine ethyl ester as a substrate. The patients were treated with furazolidine (100 mg/day for 7 days). The mean tryptic activity for 20 normal controls ranged from 12.7-27.5 μEq/min/ml, with the lower limit of normal at 8.9 μEq/min/ml. The mean tryptic activity in the giardiasis group before treatment ranged from 3.4-26.7 μEq/min/ml. There was a highly significant difference between this group and controls. Values below 8.9 μEq/min/ml were found in 10 cases (38.46%), all with diarrhea. A marked rise in tryptic activity was seen in nine patients restudied at least two weeks after completion of therapy, ranging from 10.9-26 μEq/min/ml. The difference in pre- and post-treatment values was highly significant. These data demonstrate a low tryptic activity in giardiasis; however, the mechanisms governing this activity are not clear.

6545 MEBENDAZOLE (R 17635) IN ENTEROBIASIS: A CLINICAL TRIAL IN MENTAL RETARDATES. *(E.)* Lormans, J. A. G.; Wesel, A. J. T.; Vanparus, O. F. (Inst. Ment. Retardates 't Honk, Veldhoven, The Netherlands). *Chemotherapy* 21(3/4):255-260, 1975.

The efficacy of mebendazole against enterobiasis was tested on 150 institutionalized mental retardates (age range, 5-25 yr). At the start of the trial all patients swallowed a tablet containing 100 mg of mebendazole. Another tablet was administered to all patients 2 weeks and 1, 2, and 3 months later. Further treatment with one tablet was given to 19 patients, 4 and 5 months after the start of the trial. The presence of *E. vermicularis* was checked with cellophane adhesive tapes from the anal skin and from the right-hand fingertips before the trial and after 1, 3 and 6 months. Of 150 subjects, 94 were positive before the trial. At the first post-treatment examination, all patients were negative. However, 12% were eventually reinfected. No side effects were observed or reported. These data confirm the efficacy of mebendazole in the treatment of oxyuriasis, and its lack of adverse effects.

6546 WHIPWORM DYSENTERY. *(E.)* McCaffery, T. D.; (Louisiana State Univ. Sch. Med., New Orleans). *Gastrointest. Endosc.* 21(4):172-173, 1975.

A rare case of non-inflammatory bowel disease due to whipworm infection was presented. A black female of 16 yr presented with vomiting and diarrhea of six weeks duration. Physical examination revealed evidence of dehydration. The abdomen was flat, nontender, and negative for palpable masses. Stools were positive for occult blood, and revealed *Trichuris trichiura* ova (450/direct smear) and *Entameba histolytica* trophozoites. Proctoscopy revealed massive numbers of worms attached to a friable mucosa. Colonoscopy to 40 cm revealed minimum disruption of the mucosa proximal to the friable portion, but massive numbers of worms were seen throughout. Hexylresorcinol (0.2%) retention enemas and tetracycline (one g daily for 10 days) gradually reduced egg count to 79/direct smear. *E. histolytica* infection was no longer evident on multiple follow-ups, and the dysentery cleared. No conclusions are drawn.

6547 ALTERATIONS IN NORMAL ESOPHAGEAL MOTILITY IN PATIENTS WITH CHAGAS' DISEASE. *(E.)* Csendes, A.; Strauszer, T.; Uribe, P. (Hosp. J. J. Aguirre, Santiago, Chile). *Am. J. Dig. Dis.* 20(5): 437-442, 1975.

Patients with Chagas' disease but with no esophageal involvement were followed-up to determine whether there was progression toward achalasia. Diagnosis of chronic Chagas' disease in the eight patients was based on visceral compromise (megacolon and heart A-V block), positive hemagglutination, and positive complement binding serologic tests. For radiologic examination, 4-6 spot films were taken of the esophagus and esophogastric junction in each case. The maximal internal diameter of the middle third of the esophagus and the esophogastric junction were reviewed independently by two of the authors and expressed in mm. For manometric studies, an assembly of two or three catheters was introduced through the nose or mouth up to the fundus of the stomach, and the sensors were withdrawn in one cm increments. In five patients without heart disease, 2.5 mg betamethylcoline was injected s.c. and a second pull-through was performed after 20-30 min. Relaxation was recorded as 100% when sphincter and fundic pressures were equal, and as 0% when no relaxation occurred. Follow-up after the first study ranged from 24-87 months later. The radiological studies showed no significant change other than an increase in internal esophageal diameter. Resting gastroesophageal sphincter pressure did not change significantly after a mean follow-up of 64 months. Mean sphincter length was 3.20 cm in the first study and 3.05 in the second. There was complete sphincter relaxation in five patients and partial relaxation in one in the first study. In the second study, two additional patients showed partial relaxation. Mean resting intraesophageal pressure was -3.2 mm Hg in the first study and -3.5 mm Hg in the second. Mean peristaltic wave amplitude showed an increase during the second study, but wave duration did not vary. In the first study, 76% of the waves were peristaltic compared to 89% in the second. After injection of

betamethylcoline, three patients showed a significant rise in gastroesophageal sphincter pressure; two showed no increase. It is concluded that there is no direct link between the esophageal motility of achalasia and that of Chagas' disease with respect to gastroesophageal sphincter pressure response and betamethylcoline-induced spontaneous activity.

6548 INTESTINAL MALABSORPTION IN ACUTE INFECTION WITH Strongyloides stercoralis. *(E.)* O'Brien, W.; (R. Army Med. Coll., London, England). *Trans. R. Soc. Trop. Med. Hyg.* 69(1):69-77, 1975.

An acute malabsorption syndrome, apparently resulting from early *Strongyloides stercoralis* infection, was described. The patients were seven infantry soldiers stationed in Borneo. Of them, six presented with acute diarrhea with liquid stools. The seventh presented with massive eosinophilia and associated respiratory symptoms, but developed diarrhea within four days of admission to the hospital. After admission, all were passing 4-6 loose, foul stools with a frothy appearance daily. One had abdominal colic, three had had a transient rash 2 weeks before the diarrhea, and two had had episodes of coughing, breathlessness and wheezing about the same time. Intestinal function tests showed severe steatorrhea (25-42 g/day) to be present in three cases, and moderate to mild (7.0-13 g/day) in four cases. The D-xylose test was normal, but absorption of vitamin B_{12} with intrinsic factor was subnormal in four (8.5-25%). Barium meal examination showed thickened traverse mucosal folds in two. Jejunal biopsy specimens showed a pattern of "fingers" and/or "leaves" in six, and "leaves" and ridges in one. Microscopically, the ridges showed partial villous atrophy. The villi were swollen and the vascular pattern was indistinct. Intestinal mucosa was edematous in four. In two there was heavy eosinophil infiltration. Two patients were mildly anemic. The patient with partial villous atrophy had a hemoglobin concentration of 11.8/100 ml, the marrow was megaloblastic, and serum folate was 1.6 ng/ml (it was 2-5.9 ng/ml in the other six). All showed considerable eosinophilia (over 20,000/mm^3 in two, and over 6000/mm^3 in another three). Rhabditiform larvae of *S. stercoralis* were found in the stools of one patient and in the duodenal aspirate of a second. Hookworm ova were found in these two and in three other patients. Three were treated with bephenium hydroxynaphthoate with no effect, and one received tetrachlorethylene without effect. All responded well to dithiazanine treatment. In one patient B_{12} malabsorption persisted, but returned to normal after a week of aureomycin therapy. It is concluded that the malabsorption may have been due to a hypersensitive reaction to the worms, with histamine release from intestinal mucosa mast cells.

6549 PRECIPITATING ANTIBODIES AGAINST Entamoeba histolytica IN LIVER PUS: A PRELIMINARY COMMUNICATION. *(E.)* Mahajan, R. C.; Ganguly, N. K.; Chitkara, N. L.; Dutta, D. V.; Chhuyyani, P. N. (Postgrad. Inst. Med. Educ. Res., Chandigarh, India). *Indian J. Med. Res.* 63(2):229-231, 1975.

The presence of precipitating antibodies against amebic antigen in liver pus was investigated and its titers were compared with those in serum. Pus samples were obtained from 15 cases of amebic liver abscess. Amebic immunofluorescence and indirect hemagglutination tests were positive in all cases. In each case, 20 ml of aspirate was centrifuged. The resulting supernatant and a serum sample from each patient were tested by counterimmunoelectrophoresis. Of the patients, 11 (73.3%) tested positive for amebiasis (using pus) and 14 (93.3%) tested positive using serum. Antibody titers in the serum specimens (10 were from 1:4 to 1.8) were always higher than those in the pus (nine, neat only; three 1:4). It is concluded that liver pus aspirate can be a diagnostic adjunct in hepatic amebiasis cases.

6550 DETECTION BY IMMUNOFLUORESCENCE OF ANTIBODIES TO PARASITIC AGENTS. USE OF CLASS-SPECIFIC CONJUGATES. *(E.)* Huldt, G.; Ljungstrom, I.; Aust-Kettis, A. (Natl. Bacteriol. Lab., Stockholm, Sweden). *Ann. N.Y. Acad. Sci.* 254:304-314, 1975.

Class-specific conjugates were used to study the immunoglobin (Ig) response to parasitic agents in human sera. Sera from patients with toxoplasmosis, trichinosis, South European echinococcosis, and schistosomiasis were examined by immunofluorescence (IF) using class-specific conjugates. The conjugates were also examined for Ig class specificity with *Toxoplasma* antigen, and were tested by IF for direct reactivity with each parasite. Sixteen of the 18 conjugates tested showed Ig class specificity and none reacted directly with any of the parasitic antigens. For each parasite-antibody system, chessboard titrations yielded optimum working dilutions for each conjugate; these titrations gave somewhat different results with the same conjugate in different parasite systems. In 5 infants, congenital toxoplasmosis was diagnosed by the presence of IgM antibodies in the cord blood or serum taken a few days after birth; in these cases IgM was demonstrated over periods of 2-4 months. Conversely, specific IgM antibodies were not detectable in sera taken from 3- to 12-month-old children with the congenital disease. Long-term follow-up studies of patients with acquired toxoplasmosis and trichonosis revealed demonstrable IgM and/or IgA antibodies in the early stage of infection (0-1 month). In toxoplasmosis, the IgA antibodies disappeared before the IgM antibodies, and in both infections, the eventual decrease in specific IgM antibody was associated with a rise in specific IgG antibody titer. Among 103 sera from patients with schistosomiasis, all were positive for IgG antibodies, 34% had IgM antibodies, and 6% had IgA antibodies; many of these patients had little or no clinical symptoms. In serum samples from Scandinavian Lapps with echinococcosis but no demonstrable circulating antibodies, antigenic stimulation was followed by a secondary antibody response to antibodies of the IgM and/or IgA classes. The results indicate that the use of IF for the detection of class-specific immunoglobulin response to parasitic agents can provide a reasonably specific and reproducible test if performed under optimal conditions;

the demonstration of specific IgM and/or IgA antibodies by this test probably indicates recent antigenic stimulation.

6551 COMPARISON OF METRONIDAZOLE AND CHLOROQUINE FOR THE TREATMENT OF AMOEBIC LIVER ABSCESS. A CONTROLLED TRIAL. *(E.)* Cohen, H. G.; Reynolds, T. B. (Univ. South. California Sch. Med., Los Angeles). *Gastroenterology* 69(1):35-41, 1975.

Chloroquine and metronidazole were compared in a randomized trial for the treatment of amebic liver abscess in 36 patients. An additional 30 patients were treated without randomization. The total patient population of 66 individuals was composed of 50 males and 16 females, aged 4 to 74 yr. Chloroquine was administered at a dose level of 500 mg daily for 10 weeks. Metronidazole was given in a dose of 750 mg, 3 times/day for 10 days. Criteria for the diagnosis of amebic abscess included: a suitable clinical picture, filling defect on hepatic scan, high titer of antibody of *Entamoeba histolytica* by indirect hemagglutination, and eventual complete recovery with treatment or appropriate findings on autopsy examination. Treatment failure was defined as illness that persisted beyond 10 days or recurred after that time. Such failure occurred in one out of 28 patients treated with chloroquine and in two out of 36 treated with metronidazole. In two patients who died it was difficult to assess the results of drug therapy. All three failures had unusually large filling defects on scan, and all three showed some improvement with treatment so that pyogenic abscess seemed unlikely. It is concluded that both drugs are highly effective in the treatment of amebic liver abscess.

6552 INTESTINAL NECROSIS OF THE AREA SUPPLIED BY THE SUPERIOR MESENTERIC ARTERY ASSOCIATED WITH COLONIC AND HEPATIC AMEBIASIS. *(Fr.)* Seror, J.; Weill-Bousson, M.; Jaeck, D.; Thomas, M.; Schmitt, J. C.; Korucu, B. (Univ. Hosp. Cent., Strasbourg, France). *Chirurgie* 100(12):884-890, 1974.

6553 KALA AZAR IN ADULTS IN WESTERN FRANCE FOR THE LAST 15 YEARS. *(Fr.)* Hany, Y.; Allannic, H.; Vivien, P. (Pontchaillou Hosp., Rennes, France). *Bull. Soc. Pathol. Exot.* 67(5):503-512, 1974.

6554 EOSINOPHILIC GRANULOMA OF THE COLON CAUSED BY LARVAE OF THE NEMATODE Anisakidae. *(Fr.)* Doby, J. M.; Le Masson, J. M.; Babin, P. (Fac. Med., Rennes, France). *Bull. Soc. Pathol. Exot.* 67(5):522-535, 1974.

6555 A CASE OF PERITONEAL AND PULMONARY SCHISTOSOMIASIS CAUSED BY Schistosoma haematobium. *(Fr.)* Boulay, J.; Pieron, R.; Mafart, Y.; Van Den Akker, M.; Favre, M.; Lancastre, F.; Oustrieres, G.; Andre, J. (Tenon Hosp., Paris, France). *Ann Gastroenterol. Hepatol.* 10(6):475-482, 1974.

6556 LIVER HYDATIDOSIS. EXPERIENCE OF 47 CASES. *(Sp.)* Vilaseca, J.; Vidal Pla, R.; Caralps, A.; Bacardi, R.; Vidal, M. T.; Rius, J. M. (Univ. Barcelona, Fac. Med., Spain). *Rev. Clin. Esp.* 134(3):271-278, 1974.

6557 COMMON BILE DUCT INFECTION WITH Fasciola hepatica: DESCRIPTION OF Fasciola IN HUMANS. *(Ger.)* Anghern, W.; Wegmann, T. (Canton. Hosp., Gallen, Switzerland). *Schweiz. Med. Wochenschr.* 105(22):715-718, 1975.

6558 CLINICAL PROBLEMS WITH AMEBIASIS. *(Ger.)* Stahel, P.; Aeberhard, P.; Pedrinis, E.; Halter, F. (Univ. Clin., Bern, Switzerland). *Schweiz. Med. Wochenschr.* 105(22):709-714, 1975.

6559 LARVAL NEMATODES OF FAMILY ANISAKIDAE (NEMATODA) IN THE NORTHERN SEA OF JAPAN. AS A CAUSATIVE AGENT OF EOSINOPHILIC PHLEGMONE OR GRANULOMA IN THE HUMAN GASTRO-INTESTINAL TRACT. *(E.)* Shiraki, T. (Niigata Univ. Sch. Med., Japan). *Acta Med. Biol.* 22(2):57-98, 1974.

6560 CIRCULATING IMMUNE COMPLEXES IN SCHISTOSOMIASIS DUE TO Shistosoma mansoni. *(E.)* Phillips, T. M.; Draper, C. C. (London Sch. Hygiene Trop. Med., England). *Br. Med. J.* 2(5969):476-477, 1975.

6561 PATHOGENIC MECHANISMS NOT OPERATING IN Eimeria necatrix INFECTIONS. *(E.)* Ryley, J. F. (Imperial Chem. Indust. Ltd., Macclesfield, England). *Z. Parasitenk.* 45(3):269-279, 1975.

6562 HOOKWORM INFECTION AND ANAEMIA. *(E.)* Gilles, H. M. (Liverpool Sch. Trop. Med., England). *Trop. Doct.* 5(2):51-55, 1975.

6563 ULTRASTRUCTURAL LOCALIZATION OF ACID PHOSPHATASE IN Entamoeba histolytica AND Entamoeba Gingivalis. *(E.)* Cho, K. M.; Cha, H. Y.; Soh, C. T. (Inst. Trop. Med., Seoul, Korea). *Yonsei Rep Trop. Med.* 4(1):5-18, 1973.

6564 DISTRIBUTION OF ^{131}I-RISA IN THE INTESTINAL EPITHELIA OF PARASITE INFESTED ANIMALS. *(E.)* Soh, C. T.; Kim, S. J. (Inst. Trop. Med., Yonsei Univ., Seoul, Korea). *Yonsei Rep. Trop. Med.* 4(1):19-26, 1973.

6565 CHANGES OF INTESTINAL MUCOUS MEMBRANE OF DOG WITH REFERENCE TO THE IMMUNOLOGICAL RESPONSE TO PARASITE INFESTATION. *(E.)* Soh, C. T.; Kim, S. J. (Coll. Med., Yonsei Univ., Seoul, Korea). *Yonsei Rep. Trop. Med.* 4(1):27-36, 1973.

6566 ANTIGENIC COMPARISON OF Entamoeba histolytica STRAINS BY INDIRECT FLUORESCENT ANTIBODY TEST (IFAT). *(E.)* Chung, P. R.; Chang, J. K.; Soh, C. T. (Coll. Med., Yonsei Univ., Seoul, Korea). *Yonsei Rep. Trop. Med.* 4(1):37-40, 1973.

6567 PREVALENCE OF INTESTINAL PARASITES IN ULLUNG-DO ISLAND. *(E.)* Cho, K. M.; Chang, J. K.; Chang, S. J.; Rhee, Y. S. (Inst. Trop. Med., Yonsei Univ., Seoul, Korea). *Yonsei Rep. Trop. Med.* 4(1):50-58, 1973.

6568 CONTROL OF SOIL-TRANSMITTED HELMINTHS IN KOREA. A CASE REPORT. *(E.)* Soh, C. T.; (Inst. Trop. Med., Yonsei Univ., Seoul, Korea). *Yonsei Rep. Trop. Med.* 4(1):102-125, 1973.

6569 THE SYNTHESIS OF SERUM PROTEINS IN MICE INFECTED WITH Schistosoma mansoni. *(E.)* Brown, R. A.; Knight, W. B. (Puerto Rico Nucl. Cent., Caparra Heights Stat., San Juan). *Rev. Lat. Am. Microbiol.* 16(3):169-176, 1974.

6570 INTRABILIARY RUPTURE OF HYDATID CYST OF THE LIVER. (REVIEW OF 16 CASES). *(E.)* Aletras, H.; Katsohis, C.; Aidonopoulos, A. (Dep. Surg., Univ. Thessaloniki, Greece). *Bull. Soc. Int. Chir.* 33(5-6):490-492, 1974.

6571 RECURRENT AMEBIC ABSCESS OF THE LIVER. *(E.)* Jenkinson, S. G.; Hargrove, Jr., M. D. (Louisiana State Univ. Med. Cent., Shreveport). *J. Am. Med. Assoc.* 232(3):277-278, 1975.

6572 COMMON INTESTINAL PARASITIC INFECTIONS IN THE SCHOOL-AGE POPULATION. *(E.)* Allensworth, D. D. (Dep. Allied Health Sci., Kent State Univ., Ohio). *J. Sch. Health* 45(6):331-338, 1975.

6573 THE EPIDEMIOLOGY OF COMMON INTESTINAL HELMINTHS. *(E.)* Most, H. (Coll. Med., New York Univ., N.Y.). *J. Sch. Health* 45(6):343-345, 1975.

6574 TREATMENT OF INTESTINAL ROUNDWORM INFECTIONS. *(E.)* Shookhoff, H. B. (Dep. Health, New York, N.Y.). *J. Sch. Health* 45(6):339-342, 1975.

6575 ACUTE AMOEBIC NECROSIS OF BOWEL. *(E.)* Sharma, B. D.; Chooramani, S.; Agarwal, A. K. (S. N. Med. Coll., Agra, India). *Am. J. Proctol.* 26(3):57-62, 1975.

6576 ANTIBIOTIC G-418, A NEW Micromonospora-PRODUCED AMINOGLYCOSIDE WITH ACTIVITY AGAINST PROTOZOA AND HELMINTHS: ANTIPARASITIC ACTIVITY. *(E.)* Loebenberg, D.; Counelis, M.; Waitz, J. A. (Schering Corp., Chemother. Dep., Bloomfield, N.J.). *Antimicrob. Agents Chemother.* 7(6):811-815, 1975.

See also, 5235, 5677, 5983, 6007, 6158.

GENERAL

Regional Enteritis

6577 CROHN'S DISEASE OF THE APPENDIX. PRESENTATION OF A CASE WITH REVIEW OF THE LITERATURE. *(E.)* Weiss, Y.; Durst, A. L. (Cent. Emek Hosp., Afula, Israel). *Am. J. Gastroenterol.* 63(4): 333-339, 1975.

An obese 26-yr-old male was admitted as an emergency case with a 6-day history of lower abdominal pain, which irradiated into both flanks and was initially mild. On the fifth day, the pain suddenly increased and his temperature rose to 39.9 C. On admission the patient showed marked direct and rebound tenderness in the right lower quadrant with muscle guarding. An ill-defined mass was palpated in the right iliac fossa. Diagnosis of acute appendicitis was made. At operation a large retrocecal mass was found very close to the right common iliac artery. The mass contained the gangrenous appendix and pus. The appendix was removed without burying the stump and a Penrose drain was left in the abscess cavity. The terminal ileum showed no pathological changes. On the basis of this case and many cited from the literature it is concluded that a surgeon should not hesitate to remove the appendix in the presence of regional enteritis for two reasons. First, the appendix may become the site of Crohn's disease, and second, patients with Crohn's disease may occasionally develop acute appendicitis. Indeed, all cases of Crohn's disease of the appendix showed acute appendicitis superimposed on the granulomatous lesion. Since in a patient with known Crohn's disease, the clinical picture may be interpreted as an acute exacerbation of the basic disease, one may be tempted to procrastinate, thus exposing the patient to possible complications.

6578 METRONIDAZOLE FOR CROHN'S DISEASE. *(E.)* Ursing, B.; Kamme, C. (Univ. Hosp., Lund, Sweden). *Lancet* 1(7910):775-777, 1975.

The use of metronidazole in the treatment of Crohn's disease was investigated in 5 patients (2 men and 3 women, aged 8-26 yr) who differed in respect to

duration of disease (4 months to 10 yr), symptoms (diarrhea, abdominal pain, fever, anorexia, weight loss, and sideropenic anemia), and intestinal involvement. The response to metronidazole was the same in 4 of the 5 patients. The patients received a high dose of metronidazole (up to 40 mg/kg/day) which was later reduced to about 30 mg/kg/day. Even though they received the same doses, serum metronidazole levels varied considerably from one patient to another, ranging from 5-35 µg/ml at 2-3 hr after administration of metronidazole to 2-20 µg/ml at 8 hr after drug administration. The drug-related symptoms of nausea, vertigo, and tiredness developed in all patients. Two patients developed paresthesia and hyperesthesia. Neurological examinations showed no abnormalities. Liver function tests and blood picture were normal. All side effects ceased after reduction of the dose. In response to the drug, abdominal pain diminished and defecation became less frequent. The patients gained weight and tolerated a normal diet. In one case, withdrawal of the drug caused a return of symptoms which again disappeared on reinstitution of metronidazole. In one patient, the response to metronidazole was obscure: no improvement was noted until 4 months of metronidazole treatment, and corticosteroids were reintroduced. This patient had higher serum metronidazole levels than those who showed a rapid improvement on the same dose. The response to this drug suggests that anaerobes are involved in the pathogenesis of Crohn's disease. The return to normal of the serum proteins suggested that treatment suppressed the inflammatory processes of the intestines. Of an additional 12 patients, most of them on about 20 mg/kg/day, 9 have shown a rapid clinical improvement, indicating that lower doses than those used in this study may be successful.

6579 CLINICAL MANIFESTATIONS OF CROHN'S DISEASE IN CHILDREN AND ADOLESCENTS. *(E.)* Burbige, E. J.; Huang, S. S.; Bayless, T. M. (Johns Hopkins Univ. Sch. Med., Baltimore, Md.). *Pediatrics* 55(6):866-871, 1975.

The clinical features of 58 patients (37 boys and 21 girls younger than 15 yr of age) with Crohn's disease of the small intestine and/or of the colon are summarized with emphasis on the prominence of extraintestinal manifestations. Nine patients had familial incidence of Crohn's disease or ulcerative colitis. The sites of involvement for these patients included: ileum, 18; ileum-right colon, 12; jejunum-ileum, nine; colon only, eight; duodenum-jejunum-ileum, five; jejunun-ileum-right colon, one; ileum-left colon, one; duodenum, one; and acute ileitis, three. Clinical manifestations included: abdominal pain, 86%; fever, 83%; diarrhea, 72%; growth retardation, 30%; joints, 25%; anorectal disease, 24%; bleeding, 14%; and rash, erythema nodosum, 10%. The average delay in obtaining the correct diagnosis was 13.7 months and ranged from 1 month to 7 yr. The site of the bowel involved was related to the length of the delay. Laboratory results included: increased ESR, 84%; increased WBC, 70%; decreased serum albumin, 64%; hypochromic anemia, 50%; and gastrointestinal blood loss, 36%. The results suggest that the symptoms and signs of Crohn's disease in children are often subtle and generally nonspecific. Crohn's disease should be considered a differential diagnosis of children with prolonged fever or arthralgia even without obvious gastrointestinal complaints. Of the 58 children in this study, 22 were originally thought to have diseases outside the gastrointestinal tract. Proctosigmoidoscopy, barium enema, and small-bowel series are the most useful diagnostic procedures. It is felt that an awareness of the manifestations of Crohn's disease in children as well as a high index of suspicion should shorten the delay before this diagnosis is established.

6580 LEUKOCYTE MIGRATION TEST IN CROHN'S DISEASE, ULCERATIVE COLITIS, AND ANKYLOSING SPONDYLITIS USING CROHN'S COLON HOMOGENATE, MITOCHONDRIAL, AND MICROSOMAL FRACTIONS. *(E.)* Williams, M. J.; Richens, E. R.; Gough, K. R.; Ancill, R. J. (R. United Hosp., Bath, England). *Am. J. Dig. Dis.* 20(5):425-429, 1975.

Leukocytes from 33 patients with Crohn's disease (20 females and 13 males, aged 16-66 yr), 20 patients with ulcerative colitis (15 females and 5 males, aged 16-71 yr) and 20 patients with ankylosing spondylitis (six females and 14 males, aged 16-53 yr) were tested for evidence of abnormal migration in the presence of preparations of colon from a patient with Crohn's disease. In the 33 patients with Crohn's disease, the disease was confined to the large bowel in nine, the small bowel in eight, and involved both large and small intestines in 16. None of the patients studied was undergoing treatment with immunosuppressive drugs. The same test was later performed with leukocytes from 12 healthy controls. Antigen preparations used were colon mucosal homogenate, and colon mucosal mitochondrial and microsomal fractions. The results of this study indicate that a high proportion of Crohn's disease patients have circulating lymphocytes sensitized to constituents of the diseased colonic mucosa. There are also similarities of cellular immunity between Crohn's disease and ulcerative colitis. Lymphocytes from patients with both diseases showed equal cytotoxicity to autologous and allogeneic colon cells. This phenomenon is most marked when the antigens used are the mitochondrial and microsomal fractions of the colonic mucosa. In the ulcerative colitis group, however, there was no evidence of cellular hypersensitivity to these subcellular fractions, and only minimal hypersensitivity to colonic mucosal homogenate. Therefore, this test seems to differentiate between the two diseases. Of the patients with ankylosing spondylitis, one also had Crohn's disease. With each of the three antigens used, a high percentage of patients showed stimulation of leukocyte migration. It is suggested that this may reflect weak sensitization to the antigens as opposed to the strong sensitization manifested by inhibition obtained in the Crohn's disease group. These facts may represent the opposite ends of a hypersensitivity spectrum to an antigen of common significance in the etiology or perpetuation of the disease.

6581 SOME SERUM PROTEIN STUDIES IN CROHN'S DISEASE AND ULCERATIVE COLITIS. *(E.)* Sandor, G.; Levy, E.; Malafosse, M.; Huguet, C.; Huguier, M.; Loygue, J. (Pasteur Inst., Paris, France). *Res. Commun. Chem. Pathol. Pharmacol.* 11(1):129-145, 1975.

In studying the pathogenetic factors involved in the clinical picture for Crohn's disease and ulcerative colitis, 81 determinations were carried out on about 60 cases of Crohn's disease and 73 determinations on about 50 cases of ulcerative colitis. Nine individual serum proteins were evaluated by the immunochemical radial diffusion technique: albumin, transferrin, orosomucoid, ceruloplasmin, beta-1-C-globulin, alpha-2-M-globulin, IgG, IgM and IgA. An increase in IgA, isolated or associated with that of IgM, indicated Crohn's disease; an isolated increase in IgM indicated ulcerative colitis. A significantly increased ceruloplasmin level indicated Crohn's disease; no significant immunoglobulin increase indicated ulcerative colitis. When at least two of the three major immunoglobulins increased, Crohn's disease was indicated; significantly diminished alpha-2-M-globulin indicated ulcerative colitis. An absolute albumin value of less than 20% of normal indicated Crohn's disease. When orosomucoid was highly elevated and albumin was strikingly decreased, both enterites were in acutely evolutive states. The data do not favor the hypothesis of an autoimmune origin, especially for ulcerative colitis. A prevailing IgA synthesis took place in Crohn's disease in which a mostly healthy epithelium is kept intact, but IgM synthesis prevails in ulcerative colitis, in which the mucosal epithelium is broadly destroyed. The data support the theory of a virus as the causative agent for Crohn's disease and ulcerative colitis.

6582 ORAL MANIFESTATIONS OF CROHN'S DISEASE. *(E.)* Basu, M. K.; Asquith, P.; Thompson, R. A.; Cooke, W. T. (Dep. Oral Pathol., Birmingham Univ., England). *Gut* 16(4):249-254, 1975.

The incidence of oral lesions in 100 patients with Crohn's disease (38 men and 62 women) was compared to the incidence in 100 controls and in 100 patients with ulcerative colitis (42 men and 58 women). Of these 300 individuals, nine persons with Crohn's disease (six females and three males, aged 21 to 72 yr), two with ulcerative colitis, and one normal control had oral lesions. Of the Crohn's disease patients with oral lesions, eight had involvement of both the small and large intestine. Perianal disease was found in six, and one had an eczematous skin lesion. The oral lesions macroscopically and histologically resembled those seen in the gastrointestinal tract in Crohn's disease, suggesting they were due to the same disease process. However, typical sarcoid-like granulomas were not seen. Lesions in ulcerative colitis were different macroscopically and histologically. There were no significant differences in the mean values or the incidence of low levels for serum folate, B_{12}, or serum iron between Crohn's disease patients with or without oral lesions. In contrast, an increased number of patients with oral lesions had raised serum seromucoid levels and low serum albumin levels. The parotid secretion rates of IgA for healthy controls and Crohn's patients did not differ significantly. However, Crohn's patients with raised serum seromucoid and low serum albumin had significantly lower IgA secretion rates. It is suggested that in patients with active Crohn's disease, IgA plasma cells destined for the parotid gland are diverted to the site of maximum antigenic stimulation in the bowel. Then the relative lack of IgA in the mouth of such patients allows invasion of the oral mucous membrane by food or bacterial antigens in the mouth, so producing an oral lesion.

6583 A CASE OF "MILIARY" CROHN'S DISEASE. *(Ger.)* Otto, H. F.; Gebbers, J. O.; Kugler, S. (Pathol. Inst., Univ. Hamburg, Germany). *Dtsch. Med. Wochenschr.* 100(11):505-508, 1975.

6584 FAMILIAL FORMS OF CROHN'S DISEASE. *(Fr.)* Duffaut, M.; Le Tallec, Y.; Gadrat, J.; Fournie, A.; Amigues, H.; Blanc, M.; Abbal, M.; Oksman, F. (Purpan Hosp., Toulouse, France). *Cah. Med.* 15(6):305-310, 1974.

6585 ROUNDTABLE DISCUSSION: THE DIFFERENTIAL THERAPY OF CHRONIC INFLAMMATORY DISEASES OF THE LARGE INTESTINE. *(Ger.)* Demling, L.; Fahrlander, H.; Freyberger, H.; Kuhn, H. H.; Kummerle, F. (Med. Clin., Univ. Marburg, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:328-329, 1974.

6586 A COMBINED TEST FOR THE DIAGNOSIS OF DISORDERS IN GASTROINTESTINAL PHYSIOLOGY IN REGIONAL ENTERITIS. *(Ger.)* Fromm, H.; Schindler, D.; Bar, U. (Med. Coll., Hanover, Germany). *Verh. Dtsch. Ges. Inn. Med.* 80:554-556, 1974.

6587 THE LIVER IN CROHN'S DISEASE. *(Ger.)* Morl, M.; Gruter, M. (Med. Clin., Univ. Erlangen, Germany). *Munch. Med. Wochenschr.* 117(15):637-639, 1975.

6588 DIFFERENCES IN THE TREATMENT OF CROHN'S DISEASE. *(Ger.)* Morl, M. (Med. Clin., Univ. Erlangen, Germany). *Munch. Med. Wochenschr.* 117(15):639-642, 1975.

6589 TREATMENT OF CROHN'S DISEASE WITH A BALANCED, SYNTHETIC, RESIDUE-FREE DIET (VIVASORB). *(Ger.)* Koch, H.; Belohlavek, D.; Berg, G. (Med. Clin., Univ. Erlangen, Germany). *Munch. Med. Wochenschr.* 117(15):643-645, 1975.

6590 SURGERY FOR CROHN'S DISEASE. *(Ger.)* Reifferscheid, M. (Med. Fac., Rhenish-Westphalian Tech. Coll., Aachen, Germany). *Med. Klin.* 70(17):747-756, 1975.

6591 CROHN'S DISEASE AND MALIGNANT TRANSFORMATION. *(Fr.)* Chevrel, B. (St. Antoine Hosp., Paris, France). *Med. Chir. Dig.* 3(6):431-436, 1974.

6592 NON-SPECIFIC GRANULOMATOUS DUODENITIS. *(Fr.)* Godquin, B.; Callandry, L.; Garipuy, A. (no affil.). *Chirurgie* 100(12):830-836, 1974.

6593 PSEUDO-ILEOVESICAL FISTULA IN CROHN DISEASE. *(E.)* Panitch, N. M.; Henn, R. M.; Tetrud, J. W. (Harbor Gen. Hosp., Torrance, Calif.). *JAMA* 232(7):732-733, 1975.

See also, 5881, 5934, 5956, 6505.

NUMBERS FOLLOWED BY AN ASTERISK INDICATE ABSTRACTS. OTHER NUMBERS REFER TO CITATIONS.

AARON, S.
6129*
ABAKUMOV, M.M.
6419
ABBAL, M.
6584
ABDEL WAHAB, M.F.
6540
ABE, H.
5629*
ABE, K.
6162
ABE, R.
6202
ABLITSOV, N.P.
6415
ABOU-EL-MAKAREM, M.M.
5500
ABRAM, H.S.
5900
ABSOLON, K.
5731
ABUSHAKHMANOV, KH. A.
5810
ACKERMAN, N.B.
5612
ACKERMAN, S.H.
5318
ADCOCK, D.F.
5621*
ADJUKIEWICZ, A.B.
6230
ADKINS, R.B., JR.
5297*
ADRICH, Z.
5833
AEBERHARD, P.
6558
AFFRIME, M.
6330
AGAMOVA, K.A.
5750
AGARWAL, A.K.
6575
AGARWAL, B.N.
6071
AGRAWAL, N.M.
5694
AHMED, N.
6484*
AHONEN, A.
5197*, 5576*
AHREN, C.
5867*
AHUJA, H.C.
5549
AIDONOPOULOS, A.
6570
AIGINGER, P.
6109*
AIHARA, K.
5501
AILHAUD, G.
5568*
AIZAWA, I.
5322
AJEBO, M.
6142
AKASHI, M.
6072
AKIN, F.J.
5523
AKOVBIANTZ, A.
6042
ALAAMA, A.
5980
ALAUPOVIC, P.
5624*
ALBANO, O.
6373
ALBOT, G.
5882
ALEKSEENKO, A.A.
5875
ALETRAS, H.
6570
ALETTI, M.G.
5479
ALEXANDER, S.
6222*
ALEXANDER-WILLIAMS, J.
6474
ALEXIOU, D.
5815
ALFONSO, A.E.
6004
ALGERI, R.
5657
ALJAMA, P.
W5664
ALKEN, P.
5300*
ALLAN, J.
6054*
ALLANNIC, H.
6553
ALLARDYCE, R.A.
5901*
ALLBACH, G.
5561*
ALLEN, R.P.
5417*
ALLENSWORTH, D.D.M.
6572
ALTIEV, B.A.
5957
ALTMANN, G.G.
5202*, 5227
ALTMANN, H.W.
6169
ALVARADO, F.
5243*
ALVES, N.
6491
AMBARTSUMIAN, M.S.
6308
AMDRUF, E.
5849
AMDRUP, B.M.
5314
AMDRUP, E.
5850
AMIC, J.
5424
AMIGUES, H.
6584
AMMANN, R.
5824, 6042
AMONGERO, F.
6542
ANANEV, V.G.
5844
ANANEVA, K.A.
6296*
ANCILL, R.J.
6580*
ANDERS, A.
6399*
ANDERSEN, B.
6099*
ANDERSON, C.B.
6008
ANDERSON, L.G.
5025*
ANDERSON, P.J.
5634*
ANDERSON, P.R.
5299*
ANDERSON, W.
5930*
ANDERSSON, A.
5991
ANDERTON, R.L.
6358
ANDRE, J.
6497, 6555
ANDREASEN, P.B.
6078*
ANGELOPOULOS, B.
6357
ANGHERN, W.
6557
ANGULO, A.E.
5797
ANSALDI, N.
5912
ANSPACH, K.
5670
ANTIA, F.P.
5848
ANTONOWICZ, I.
5924
ANTUNES, L.J.
6053*
ANWER, M.S.
5353*
APOSTOLOV, B.G.
5874
APPUKHAMI, K.R.A.
5759
ARAI, K.
5502
ARAKAWA, T.
5432
ARAKI, E.
6131
ARANY, Y.
6159
ARCHAKOV, E.V.
6411
ARCHAKOV, I.I.
5451
ARCHIBALD, R.W.R.
6333*

AKCHIMANDRITIS, A.
6357
AKCORACI, A.
6183
AKENDT, D.
6387*
AKGYRIS, T.S.
5458
ARIANOFF, A.A.
6430, 6432
ARIANOFF, V.
6430, 6432
ARITA, T.
6162
ARIYAN, S.
6086*
ARNAUD, S.B.
5387*
ARNFRED, T.
6386*
ARNOLD, K.
5804, 5839
ARNOLD, W.
5542, 6276
ARNOUX, A.
5669
ARON, E.
6351
ARVIDSSON, B.
6479*
ASANO, G.
5502
ASARIAN, J.
6333*
ASATOUR, A.M.
5259*
ASCASO, J.
6057*
ASHBY, D.B.
5313
ASHMAN, M.
6011*
ASQUITH, P.
6582*
ASRAF, S.M.
6010
AST, E.
5414*
AST, H.
5915
ASTARITA, R.W.
5688
ATHANASOULIS, C.A.
5987
AUBREY, D.A.
5305*
AUCANT, D.
5636*
AUDIGIER, J.C.
5321
AUKEE, S.
6401*
AURANEN, A.
6404*
AURIOL, M.
6142
AUST-KETTIS, A.
6550*
AVAGNINA, P.
6355
AVGOUSTIS, A.
5853
AZARNOFF, D.L.
6317*
AZAROVA, I.V.
5766, 5773
BABA, T.
5316
BABB, R.R.
6201
BABIC, K.
5606
BABIN, P.
6554
BACARDI, R.
6556
BACH, A.
5363*
BADDELEY, R.M.
6474
BADEN, H.
6099*, 6107*
BADR-EL-DIN, M.K.
6543
BAERLOCHER, C.
5625*
BAGGENSTROSS, A.H.
6344*
BAGRATUNI, B.E.
6074
BAHODORZADEH, K.
5725
BAHUAUD, J.
5880
BAILEY, D.G.
5263
BAILLIE, T.A.
5390*
BAISOGOLOV, G.D.
5942
BAKER, D.H.
5733, 6081*, 6204
BAKER, P.G.
5923
BAKER, R.J.
5396*, 5634*
BAKHADYROV, K.B.
6454
BAKHRUSHEVA, T.S.
6412
BALABONKIN, V.G.
6268
BALALYKIN, A.S.
5775
BALASEGARAM, M.
6088*
BALLINGER, W.F.
6008
BALTAYTIS, IU. V.
5845
BALTEANU, E.
5475
BALTZER, G.
6051*
BAMFORD, D.R.
5251*
BAMONTI, F.
6194
BAN, A.
6429
BANERJEE, D.
5374*
BANKS, P.A.
6481*
BANNASKI, H.
6289*
BANNET, D.
6038
BAR, U.
6586
BARAKAT, M.Z.
5500
BARAN, M.M.
5237*
BARANOVSKII, A.L.
5971
BARBAGELATA, M.
5945
BARBERA, C.
5912
BARBEZAT, G.O.
5574*
BARCHUNOV, B.N.
5827
BARCHUNOVA, N.N.
6488
BARDACH, G.
5958
BAREICHA, I.
5564*
BARKE, R.
6030*
BARNES, S.
5632*
BARR, J.W.
5415*, 5533
BARREAU, J.
5777
BARRETO, D.G.
6525
BARRON, P.T.
5498
BARTKOWIAK, Z.
6439
BARTSCH, G.G.
5419*
BASILE, A.
5706
BASS, E.M.
5696
BASS, P.
5246*, 5269*
BASSLER, R.
6462
BASSO, N.
5734*
BASU, M.K.
6582*
BAUDRY, M.
5426
BAUDUIN, H.
5338*
BAUER, E.
5414*, 6387*

BAUER, H.
5804
BAUM, M.
6034
BAUMANN, J.C.
6178
BAUTZ, B.
5716
BAYAR, M.
5977
BAYLESS, T.M.
6579*
BEAUDOIN, J.G.
5790
BEAUDRY, C.
5751
BEAVON, J.R.G.
5454
BECK, J.D.
6398*
BECKER, H.
5879
BECKER, J.C.
5636*
BECKER, J.E.
5264
BECKER, V.
6408
BEFFAGNA, B.
5515
BEGEMANN, F.
6380*
BEGER, H.C.
6423
BEGER, H.G.
5823
BELIAEV, A.A.
6044
BELIAKOV, N.V.
5758
BELLEN, J.C.
5396*, 5634*
BELLET, H.
6366
BELLMAN, H.
5544
BELLOMI, M.
6135
BELOHLAVEK, D.
6589
BELTRANDI, E.
6181
BEMELMANS, D.H.
5508
BENACERRAF, R.
5655
BENDIRDJIAN, J.P.
5443
BENEDETTI, G.
6334*
BENEKE, G.
5422
BENNETT, P.N.
5258
BENNETT, W.M.
6346*
BERDON, W.E.
5733, 6081*, 6204
BERDOV, B.A.
5942
BERG, G.
6589
BERG, P.A.
6101*
BERGAMASCHI, R.
6459
BERGDAHL, L.
5991
BERGER, D.
5638*
BERGER, E.R.
5378*
BERGER, J.
6290*
BERK, J.E.
5994
BERKMAN, N.L.
6464
BERKUS, O.V.
5290
BERKOWITZ, D.
6007
BERNADES, G.
6377*
BERNARD, C.C.
5466
BERNE, T.V.
6247*
BERNI, G.
6354
BERNIER, J.J.
5257, 6028*
BERNSTEIN, L.H.
6007
BERSIMBAEV, R.I.
5746
BERTA, L.
6355
BERTACCINI, G.
5289, 5328
BERTHOLD, H.
6243*
BERTOLINO, A.
5372*
BERTRAMS, J.
6245*
BERTRAND, L.
6366
BESSARD, G.
5871
BESSOT, M.
5677
BEZARD, J.
5435
BHATHAL, P.S.
6331
BIANCHETTI, A.
5315
BIANCHINI, D.
5912
BIENENSTOCK, J.
5604
BIENFAIT, D.
5431
BIERNACKY, J.
6431
BIGOTTI, A.
5641
BILBAO, M.K.
5618*
BILIBIN, A.F.
6502
BILLINGTON, T.
5565*
BIRASCHI, L.M.
5315
BISCHOFF, B.
6148
BISMUTH, H.
6365
BJORKSTEN, B.
5997
BLACK, M.
6218*
BLACKSTONE, M.O.
6069
BLAIR, J.A.
5454
BLANC, M.
6584
BLANQUET, P.
5665
BLANT, M.E.
6511
BLENDIS, L.M.
6359
BLEYL, H.
6148
BLOCH, P.
6092*, 6167
BLOCK, J.A.
5497
BLOOM, G.D.
5236
BLOOMER, J.R.
6117*
BLUM, A.L.
5852
BLUMBERG, B.S.
6248*
BLUMGART, L.H.
5384*, 5465
BOBOIS, J.P.
6152
BOCKHORN, H.
5398*
BOCKMAN, D.E.
5203*
BODDY, K.
5910*
BODE, C.
5447, 6315*
BODE, J.C.
5447, 6173, 6315*
BODOVA, A.G.
6261
BOECK, P.
5418*
BOECKER, W.
6457
BOGER, M.M.
5746
BOGLIOLO, G.
5295

BOGUSCH, G.
6392*
BOHM, E.R.
5909*
BOHM, N.
5218
BOHNE, A.
5918
BOICHIK, G.K.
6372
BOIKO, Y.G.
5762
BOJANOWICZ
6076
BOLSHAKOVA, N. IA.
6513
BOLTE, J.P.
5914
BOMBECK, C.T.
5684
BONE, E.
5928*
BONE, J.
5314
BONORRIS, G.G.
5366*
BONTA, I.L.
5281
BORIES, G.
5543
BORNEMANN, W.
6420
BORON, P.
6250
BORT, J.
6057*
BORTHISTLE, B.K.
5596*
BOSMANN, H.B.
5562*
BOTERASHVILI, M.I.
5845
BOTSVADZE, E.S.
6281
BOULAY, J.
6555
BOUMA, J.M.W.
5402*
BOURDAIS, J.P.
5780
BOURGEOIS, B.
6490
BOUT, D.
6544*
BRACALE, G.
5922
BRACHTEL, D.
6130
BRAILSKY, K.
5826
BRANDBERG, A.
5867*
BRANDSBERG, M.
5314
BRANDSBURG, G.
5314
BRANT, P.C.
6335*
BRASHER, G.W.
6026*
BRATABOVA, V.
6532
BRAUN, D.
5823
BRAUN, L.
5707, 5939*
BREAM, C.A.
5621*
BRECHT, T.
5771
BRENNAN, T.G.
5940*
BRENNER, R.R.
5551
BRENSILVER, H.L.
6113*
BRICKER, R.S.
6036
BRIJS, V.A.
5872
BRITTON, D.C.
6402*
BRIZGALOV, V.E.
5951
BROCHARD, C.
6143
BROCHERIOU, C.
6142
BRODIN, E.
5567*
BRONSTEIN, H.A.
6480*
BROUWERS, H.P.
5675
BROWN, B.
5987
BROWN, C.S.
6047*
BROWN, J.C.
5273*
BROWN, P.W.
5936*
BROWN, R.A.
6569
BROWN, W.R.
5596*
BRUGGEMAN, T.M.
5303*
BRUGMANN, G.
5919
BRUHN, H.D.
6189
BRUN DEL RE, G.
6498
BRUNNER, H.
5445, 5869
BRUNSWIG, D.
6336*, 6343*
BSTEH, O.
5937*
BUBRICK, M.P.
6067
BUCHANAN, B.J.
5496
BUCHELER, E.
6166, 6396*
BUCHENAU, D.
6343*
BUCHWALD, H.
5894
BUDILLON, G.
5922
BUFFA, R.
5198*
BUFFE, M.D.
6189
BUFFONI, L.
6227
BULGAKOV, G.A.
5745
BULGAKOVA, O.S.
5681
BULL, D.M.
6314*
BUNIN, K.V.
6512
BUNIN, M.V.
6512
BUR, F.
5815
BURBIGE, E.J.
6579*
BURCHER, S.K.
6056*
BURCHINSKI, G.I.
5821
BURDELSKI, M.
5671
BURDMAN, D.
5993
BURDON, D.W.
6474
BURHENNE, H.J.
6470
BURHOLT, D.R.
5234, 5589
BURKE, J.F.
5720
BURKO, H.
5737*
BURKS, T.F.
5267*
BURLINA, A.
6310
BURNS, G.P.
5305*
BURNSWIG, D.
5397*
BUSH, M.
5635*
BUSSET, A.
6319
BUYANOV, V.M.
5775
BUYS, C.H.C.M.
5402*
BYBEE, J.W.
5233
CAHTTERJEE, S.N.
6247*
CAIRNIE, A.B.
5200*, 5592*
CALCRAFT, B.J.
5255

CALDERON, E.
6525
CALDERON, P.
5350
CALDWELL, J.H.
6480*
CALLANDRY, L.
6592
CALLET, B.
6377*
CAMERON, J.L.
6049*
CAMPBELL, D.P.
5624*
CAMPBELL, H.
6212*
CAMUSSI, G.
6295*
CANDARDJIS, G.
5656
CANDEL-MONSERRATE, I.
5439, 6177
CANO, C.
5607
CANO, J.M.
6062
CANTALLOPS, J.G.
6061
CANTALUPPI, G.
5515
CANTARERO, L.
5588
CANTOR, D.S.
5672
CAPELLA, C.
5198*
CAPORASO, N.
6180, 6285
CAPPEL, R.
6280
CAPPS, W.F., JR.
6075
CAPRILL, R.
5270*
CAPRON, A.
6544*
CAPRON, J.P.
5442
CAPRON, M.
6544*
CAPUTO, M.
6507
CARACCIOLO, P.
6535
CARALPS, A.
6556
CARAMIA, F.
6251, 6286
CARBON, M.
6313*
CARDOSO, A.
6490
CARLSON, H.C.
5863*
CARLSSON, B.
5236
CARMENA, R.
6057*
CAROLI, J.
5908*, 6448, 6453
CARPIO, N.M.
6215*
CARSWELL, F.
5910*
CARTEL, G.
6334*
CASAGRANDE, A.
5911
CASARELLA, W.J.
6081*
CASH, R.A.
6486*
CASPER, J.T.
6110*
CASS, O.W.
6102*
CASSAIGNE, A.
5472
CASTAGNA, M.
5495
CASTRO, G.A.
5285*, 5591*
CASTRO, G.A.M.
6318*
CATTO, E.
5548
CAVALLO, C.
6000
CAVARZAN, A.
5348
CECIO, A.
6180
CELLE, G.
5294
CENTONZE, M.
6373
CESARONI, R.
5667
CHA, C.J.M.
6024*
CHA, H.Y.
6563
CHA, S.
6024*
CHAFFEE, V.
5934*
CHAINUVATI, T.
6236*
CHALMERS, I.
6212*
CHANARD, J.
6288
CHANDLER, J.G.
5400*
CHANDRASEKARAN, R.
6517
CHANG, J.K.
6566, 6567
CHANG, L.W.
5395*, 5518
CHANG, M.
6197
CHANUSSOT, F.
5435
CHAPPELKA, A.R.
6022
CHAPUT, J.C.
5650
CHARBONNE, F.
5449
CHARIOT, J.
5347
CHARTERS, A.C.
5400*
CHARTIER, P.
6395*
CHAVES-CARBALLO, E.
6233
CHEESEMAN, C.I.
5573*
CHEKALIN, A.P.
6416
CHEKHLAROV, V.
5826
CHEKURISHVILI, R.K.
5952
CHELI, R.
5915
CHEN, S.
5930*
CHEN, S.T.
5596*
CHEN, T.
6328
CHENDEROVITCH, J.
5480
CHENG, C.Y.
5248*
CHENG, H.F.
5587, 5588
CHENOUDA, M.
6405*
CHEREDNICHENKO, T.V.
6261
CHERNIACK, N.S.
6345*
CHERNOVA, G.V.
6421
CHERNYI, V.A.
5646
CHERRY, W.B.
6518
CHETVERIKOV, D.A.
5437
CHEUNG, L.Y.
5207*, 5590*
CHEVALLIER, F.
5579
CHEVREL, B.
6591
CHEVREL, J.P.
6122*
CHHUTTANI, P.N.
5376*
CHHUYYANI, P.N.
6549*
CHIAMPO, L.
5608
CHIAVARINI, M.
5289
CHIRVA, V.I.
5772

CHISTOVA, M.A.
5758
CHITKARA, N.L.
5557, 6549*
CHIVARINI, M.
5328
CHO, K.M.
6563, 6567
CHOBOTAR, B.
5235
CHOE, D.S.
6519
CHOI, B.H.
5519
CHOMETTE, G.
5577, 6142
CHOORAMANI, S.
6575
CHOPRA, P.
5534, 5535
CHOWDHURY, J.R.
5978
CHRISTENSEN, G.M.
5509
CHRISTIANSEN, J.
5307*
CHRISTIEN, G.
5778
CHRISTOPHE, J.
5349, 5350
CHUNG, P.R.
6566
CHYLINSKI, J.
5833
CLARK, L.C., JR.
5223
CLARK, M.L.
5244*
CLARK, R.D.
6238*
CLASSEN, M.
5676, 5905*, 6400*
CLEMENCON, G.
5704*
CLEMENS, W.
6396*
CLIFFORD, B.
5242*
CLIFTON, S.
6331
CLINKENBEARD, K.D.
5471
CLOT, J.P.
5426, 5577
CLOWES, G.H.A., JR.
5404*
COAN, M.G.
5580
CODE, C.F.
5309*
COHEN, A.H.
6087*
COHEN, C.
6098*
COHEN, H.G.
6551*
COHEN, S.
6514
COHEN, S.E.
5860*, 5861*
COLALONGO, G.
6181
COLLINS, M.A.
5539
COLLINS, P.J.
5634*
COLONI, G.F.
5734*
COLTORTI, M.
6285, 6352
COLVIN, B.T.
6021
COMBES, B.
5411*
CONARD, J.
6167
CONCANNON, J.P.
5581
CONDON, J.R.
5859
CONN, H.O.
5393*
CONN, J.H.
5933*
CONSTANTIN-VOILLEMOT, N.
5225
CONWAY, W.D.
5453
COOK, J.A.
5369*
COOK, M.G.
6015*
COOKE, A.R.
5260, 5282
COOKE, W.J.
5362*
COOKE, W.T.
6582*
COONEY, D.R.
5570*
COOPER, J.
5212*
COOPER, J.W.
5234
COOPER, M.D.
5203*
COOPER, W.
5259
COOVER, J.A.
5369*
COPELAND, E.M.
5285*, 5591*
COPPER, J.W.
5569
COPPI, G.
6459
CORDERO, D.C.
6066
CORDIANO, C.
5713
CORMIANO, C.
5714
CORMAN, J.
6092*
CORMAN, J.L.
6080*
CORNELL, R.P.
5496
CORNIER, E.
6167
CORREA, P.
5927*
CORRIVEAU, M.
5204*
COSTA, C.D.
6490
COSTA, J.A.
5672
COSTANTINI, F.
5580
COTTIER, H.
6498
COUDOUX, P.
6038
COULAUD, J.P.
6143
COULSTON, F.
5477
COUNELIS, M.
6576
COUROUCE-PAUTY, A.M.
6279, 6288
COWEN, A.E.
5409*, 6102*
COYNE, M.J.
5366*
CRAIG, J.R.
6247*
CRAIG, R.M.
6011*
CREED, K.E.
5611
CREMER, M.
5349
CRESPI, M.
5641
CREUTZFELDT, C.
5839
CREUTZFELDT, W.
5804, 5839
CREUTZIG, H.
5680
CROCE, G.
6041
CRONHOLM, T.
5388*
CROTTE, C.
5428
CSENDES, A.
6465, 6547*
CSICSKO, J.F.
5570*
CUBILLA, A.L.
6031
CUELLO, C.
5927*
CUELLO, R.
5868*
CULLIFORD, A.
5983
CUMMINGS, M.G.
5467
CURRAN, P.F.
5256

CURRY, N.
5306*
CURWAIN, B.P.
5323
CUTHBERT, A.W.
5584
CVETANSKI, I.
5877
CYTAWA, J.
5278
CZOK, G.
5410*
CZYGAN, P.
6120*, 6124*
DA SILVA HORTA, J.
6176
DA SILVA HORTA, M.E.
6176
DABROWSKI, Z.
5776
DADOO, R.C.
5988
DAFNY, N.
5603
DAGRADI, A.E.
5980
DAIRCU, R.
5442
DALEZIOS, J.I.
5524
DALLAKI, A.
6459
DALOZE, P.
6092*
DALVI, R.R.
5491
DALY, J.W.
5530
DAM, A.P.M.V.
5752
DAMIANO, A.
W5604
DAMM, K.H.
5520
DAMMANN, H.G.
5554
DAMSGAARD-SORENSEN, P.
5687
DAN, M.E.
6082*
DANCEA, S.
6429
DANDAVINO, R.
5751
DANE, D.S.
6305
DANESE, C.
5586
DANI, R.
6053*
DANILOV, L.N.
6447
DANO, P.
6116*
DARNIS, F.
5440, 6141, 6303
DAS, K.
5978
DAS, K.M.
5978, 6023
DAS, P.K.
5534, 5535
DATTA, D.V.
5376*
DAVENPORT, H.W.
5311*
DAVID, T.J.
5724
DAVIDENKO, L.A.
5819
DAVIDOVSKAIA, N.I.
6419
DAVIDSON, C.S.
6314*
DAVIDSON, F.Z.
6219*
DAVIES, M.
5855
DAY, E.A.
5864*
DBJAY, H.C.
5880
DE AGUIAR GONZALEZ, A.R.
5917
DE ARAUJO MORAES, N.L.
6504
DE BAC, C.
6286
DE BENEDETTI, F.
6309
DE DIEGO, J.A.
5607
DE DIOS, J.F.
6062
DE GIROLAMO, G.
6180
DE GOMEZ DUMM, I.N.T.
5551
DE JONG, A.S.H.
5402*
DE JONGE, H.R.
5575*
DE LA TOUR, J.
5347
DE LUCA, V.
6507
DE MORAES-FILHO, J.P.P.
5800*
DE OLIVIERA LINS, J.C.
5669
DE PERGOLA, E.
6374
DE PRA, M.
5911
DE RITIS, G.
5907*
DE, S.
6539
DE, S.P.
6539
DE SAINT BLANQUAT, G.
5254
DE SANTIS, L.
6227
DEAN, R.H.
5900
DEBAC, C.
6251
DEBAS, H.T.
5271*
DEBELLE, R.C.
5783
DEBRAY, C.
5347, 6255
DEBRY, G.
5435
DEDERSEN, G.
5686
DEDIKOVA, L.A.
6296*, 6371
DEEG, P.
6369
DEGROOTE, J.
6144
DEI CAS L.
6175
DEICHER, H.
6275
DEITRICK, J.E.
5386*
DEL PRETE, S.
6305
DEL VECCHIO-BLANCO, C.
6180, 6285
DELACROIX, G.
6430
DELAVIERRE, P.
5780
DELEVETT, A.F.
5868*
DELMONT, J.
5582
DELORME, F.
6487
DELTENRE, M.
5349
DELVIN, E.E.
6092*
DEMBINSKI, A.
5332
DEMIN, M.D.
5646
DEMITRESCU, T.
5332, 5333
DEMLING, L.
6400*, 6585
DENARDO, G.L.
5630*
DENARDO, S.J.
5630*
DENCHEV, V.
6527, 6532
DENEZ, B.
6422
DENNIS, C.
6405*
DENT, T.L.
5856
DEPORTE, A.
6365
DERACHE, R.
5254

DEROO, J.
5419*
DEROOS, J.
5339*
DESAI, H.G.
5848
DESAIVE, C.
5959
DESCHNER, E.E.
5931*
DESGREZ, M.A.
5666
DESJEUX, J.F.
5256
DESNOYERS, P.A.
5395*
DESSANIT, J.P.
6544*
DETKY, B.
5961
DEVIVO, D.
6197
DEWULF, E.
6430, 6432
DEYHLE, P.
5824
DHAR, A.
5534, 5535
DI LUZIO, N.R.
5250*, 5369*
DI MATTEO, A.
5641
DI SILVERIO, C.
5659
DI SIMONE, A.
6180
DIARD, F.
6152
DICKERSON, J.W.T.
5456
DICKMAN, M.D.
6022
DICKSON, E.R.
6344*
DIDATO, A.
6306
DIDOLKAR, M.S.
6032
DIEDERICHSEN, H.
5700
DIEGO MARIN, J.
6170
DIETSCHY, J.M.
5358*
DILAWARI, J.B.
6013*
DIMITROVA, Z.
6532
DINI, M.
6194
DINSTL, K.
6442
DIOGUADI, N.
6302
DIORIO, A.F.
5499
DITTMANN, K.
6105*
DITTRICH, J.K.
5716
DIVERTIE, M.B.
5929*
DJACZENKO, W.
5559
DLUGOSZ, J.
6191
DMITRIENKO, V.V.
5711
DMITRIEV, G.I.
5827
DOBREV, J.
6438
DOBY, J.M.
6554
DOCKRAY, G.J.
5593*, 6483*
DODERO, M.
5294
DODGE, J.A.
5791
DOEHN, M.
5889, 6073
DOHRMANN, R.
5884
DOMANSKI, Z.
5836
DOMICICK, A.C.
5670
DOMINGO, N.
5429
DOMSCHKE, S.
5905*
DOMSCHKE, W.
5905*
DONALDSON, M.
5936*
DONATI, A.
5753
DONIACH, D.
6305
DOSSETOR, J.F.B.
5904*
DOURDOUREKAS, D.
6195
DOUTRE, L.P.
5654, 6152
DOVZHIKOV, V.I.
5944
DOYLE, J.J.
5526
DOYON, M.
5973
DRABKIN, D.L.
5463
DRAGIEV, M.
5513
DRAPER, C.C.
6560
DRASAR, B.S.
5928*
DRASKOVICOVA, M.
6529
DREILING, D.A.
5586, 5690, 6029*, 6070
DRESSLER, J.
6368
DROBROVOLSKAI, S.G.
5436
DROCHMANS, P.
5373*, 5375*
DROUET, J.
6288
DROZDOVSKAYA, G.M.
5747
DRYBURGH, J.R.
5273*
D'SOUZA, D.W.
5598*
DUBARRY, J.J.
5665
DUBÉ, P.
5718
DUBIN, J.C.
5334
DUBRU, J.M.
6493
DUCHESNE, J.
5527
DUCHEVA, S.
6532
DUCK, B.W.
6224*
DUCLUZEAU, R.
6154
DUDLEY, A.W., JR.
5518
DUDNIK, V.S.
5758
DUDRICK, S.J.
5285*, 5591*
DUFFAUT, M.
6584
DUGAN, R.E.
5529
DUJOVNE, C.A.
6317*
DUMITRU, I.F.
5475
DUMONT, M.
5431, 5442
DUNCHEVA, S.
6527
DUNLOP, E.H.
6089*
DUPONT, J.
5552
DUPUY, R.
6377*
DURR, C.
5414*
DURST, A.L.
6577*
DUSHANIN, S.A.
6372
DUTTA, D.V.
5557, 6549*
DUVAL, M.
5431
DUVEAU, D.
5972
DYBICKI, G.
6434
DYCK, W.P.
6026*

DZHARIMOV, A.Y.
5829
DZHIOEV, V.G.
6446
DZIENISZEWSKI, J.
5287*, 5337*, 5348
EARP, H.S.
5490
EASTWOOD, M.A.
6023
EBINA, Y.
5357*
ECKERT, P.
6073
EDINGTON, T.S.
5688
EDLIN, P.
6542
EDMONDS, C.J.
5799*
EDWARDS, J.A.C.
6474
EDWARDS, K.D.G.
5239*
EFSKIND, L.
5719
EGGERTH, G.
5869
EISEMAN, B.
6112*
EISENBACH, J.
6133
EISENSCHER, A.
5636*
EKELUND, G.
5932*
EKSTROM, J.
5291
EKZARKHOV, B.A.
6372
EL MACLA, G.
6003
EL-MOFTY, S.K.
5385*
ELETSKII, IU.K.
5345
EL'GORT, D.N.
6094*
ELIAS, E.G.
6032
ELLIS, F.H., JR.
5721
ELLIS, H.
5792
ELLIS-PEGLER, R.B.
6477*
EMMRICH, R.
6427
EMONS, D.
5909*
EMORY, C.
5223
ENDERLIN, F.
6397*
ENDO, H.
5316
ENDSBERGER, G.
6039
ENG, K.
5514
ENGEL, J.J.
6318*
ENGELHART, G.
5625*
ENGLUND, C.W.
5587
ENOMOTO, I.
5644
EPAND, R.M.
5452
ERB, W.
6043, 6105*
ERBE, R.
6274
ERIAN, M.
6540
ERIKSEN, P.O.
5314
ERIKSSON, H.
5390*
ERLIKSSON, H.
5388*
ERLINGER, A.
5442
ERLINGER, S.
5431
ERNST, J.V.
5235
ERTTMANN, R.R.
5520
ESAFOURI, S.
6003
ESCRIBANO, P.M.
5607
ESPOSITO, A.
6459
ESSIOUX, H.
6158
ESTEBAN, J.
W5664
ESTENSEN, R.
6091*
ESTES, L.W.
5340*
ESTEVENON, J.P.
6050*
ESTHER, L.
5245*
ETANI, N.
5629*
EVANGELISTI, G.B.
6456
EVANS, D.C.
5308*
EVTUSHENKO, A.YA.
5438
EWE, K.
6509
EWERS, H.R.
5675
FABIAN, W.
5879
FAGGIOLI, M.
6456
FAGNIEZ, P.L.
5577
FAHRLANDER, H.
5625*, 6585
FAIVRE, J.
6154
FAIVRE, M.
6154
FALISEVAC, V.
6253
FALKSON, G.
6196
FAN, V.S.
5859
FARBER, J.
5385*
FARELLO, G.A.
5713, 5714
FARID, A.
6540
FARKAS, A.
6443
FAROOQ, O.
5271*
FATTAKHOV, K.S.
5873
FAVRE, M.
6555
FAYS, J.
5677
FAZZINI, G.
5805
FEDOTOV, V.V.
6261
FEHMERS, M.C.O.
5921
FEIEREIS, H.
6017
FEIFEL, G.
5660
FEIGIN, R.D.
6197
FELDMAN, E.B.
5248*
FELDMAN, S.
6011*
FELLNER, J.
5580
FELLOWS, R.A.
6204
FELSENFELD, O.
5694
FELSHER, B.F.
6215*
FERLA, G.F.
6135
FERNANDEZ, M.
6465
FERRANDO, J.
6057*
FERRET, O.
6304
FESENKO, I.P.
6155
FETISOV, V.A.
5221, 5774
FEURLE, G.
5838
FICHERA, G.
6135

FIELDING, L.P.
5323
FIESSINGER, J.N.
5650
FIGARELLA, C.
6050*
FIGGE, H.
6342*
FILHO, O.
6490
FILIPESCU, G.
5475
FILIPPINI, L.
5663, 5712
FILKINS, J.P.
5496
FILLASTRE, J.P.
5443
FINGEROTH, R.J.
5393*
FIORE, M.R.
6507
FIORENTINI, C.
6349*
FIORENTINI, G.P.
6181
FIRSOV, V.D.
5951
FISCHER, B.
5444
FISCHER, J.E.
5467
FISHER, D.J.H.
6021
FISHER, M.M.
5408*, 5457
FITTS, W.T., JR.
5782
FITZGERALD, P.A.
5989
FITZGERALD, P.J.
6031
FIXA, B.
5760
FLECKENSTEIN, P.
5686
FLEISCHER, K.
5343*
FLEISCHMANN, R.
6150
FLEMING, C.R.
5863*, 6344*
FLEURY, J.
6490
FLUEGEL, H.
5631*
FLUTE, P.T.
6238*
FLYE, M.W.
6390*
FODISCH, H.J.
6410, 6455
FODOR, O.
6429
FOMENDO, T.L.
6156
FOMINA, A.A.
5818
FONT, R.
6083*
FONT, R.G.
5694
FORAFONOVA, L.N.
6058
FOREMNY, Z.
6016*
FORNES, J.
6057*
FORREST, J.A.H.
6012*
FORSSMAN, O.
5633*
FORSTER, E.
6290*
FORSTER, F.J.
6133
FORTNER, J.G.
6516
FOSTER, J.H.
5297*
FOUCHER, B.
5443
FOUET, P.
6422
FOURNIE, A.
6584
FOWLER, P.D.
6222*
FRACASTORO, G.
5713, 5714
FRANCAVILLA, A.
6373
FRANCHINI, A.
5963
FRANCO, D.
6365
FRANK, K.H.
6271
FRANKEN, F.H.
6257
FRANKHUIJZEN, A.L.
5281, 5284
FREANT, L.
5731
FREEDMAN, P.
6535
FRESCO, G.
5294
FREW, E.M.S.
5854
FREXINOS, J.
6159
FREYBERGER, H.
6585
FRIEDE, J.
5973
FRIEDMAN, E.M.
6542
FRIERI, G.
5270*
FRIGO, G.M.
5258
FROMM, H.
6586
FROMMHOLD, H.
6383*
FROMMHOLD, W.
6383*
FROTZ, H.
6165
FRUCHART, J.C.
6320
FUCHS, K.F.
5544
FUCHS, W.A.
6210*
FUCIK, M.
5809
FUJII, M.
5262
FUJIMOTO, A.
6040
FUJITA, M.
6524
FUKUI, N.
5357*
FULTON, R.L.
6129*
FULTON, T.T.
6097*
FUMAGALLI, G.
5215*
FUNCK-BRENTANO, J.L.
6288
FUNDORA TAYOR, I.
6160
FUNG, K.
5718
FUNOVICS, J.
5445
FUNOVICS, J.M.
5467
FURNELLE, J.
5350
FURNIVAL, C.M.
5384*
FURUYA, A.
5262
FUTORIAN, E.S.
6445
FUX, H.D.
6185
GADRAT, J.
6584
GADSHIEVA, Z.M.
5219
GADZHIEV, A.S.
5844
GAFFEY, T.A.
5862*
GAGINELLA, T.
5246*
GAGINELLA, T.S.
5269*
GAGLIARDI, C.
5922
GAIA, E.
6041
GAIDANO, G.
6355
GAIDICHUK, S.T.
5828
GAINER, R.B.
5930*

GALAEVA, S.S.
6488
GALDDEN, J.B.
6518
GALETSKAIA, T.M.
5818, 5821
GALIAN, A.
6028*
GALLO, G.A.
5632*
GALMARINI, D.
5515
GALPERIN, B.L.
6263
GALZIGNA, L.
6310
GAMMILL, S.L.
6083*
GANCHENKO, L.I.
5763
GANGULY, N.K.
6549*
GARACI, E.
5559
GARAGNANI, A.
6456
GARCIA BENGOECHEA, M.
6138
GARDNER, B.
6405*
GARIPUY, A.
6592
GARNIER, H.
5577
GARRIDO, H.
5607
GARRISON, J.C.
5470
GARTNER, S.L.
5507
GASTEVA, S.V.
5437
GATHMANN, H.
6211*
GAUCHER, P.
5677
GAYLARDE, P.M.
6230
GAZZARD, B.G.
6089*, 6238*
GEBBERS, J.O.
6583
GELLER, L.I.
5681
GELOV, N.
5825
GENOVA, G.
6121*
GEOFFROY, H.
6038
GEORGH, D.
6529
GEORGE, P.K.
6481*
GERBER, G.B.
5419*
GERHARDT, H.
6293*
GERLOVIN, E.SH.
5346
GEROLAMI, A.
5428
GEROULANOS, S.
5878
GEWERTZ, B.L.
6203
GHOSE, C.R.
5610
GHOSH, A.K.
5610
GIAMPAGLIA, G.
5805
GIANNELLA, R.A.
5205*
GIARDION, R.
5963
GIBB, S.P.
5721
GIEZAAI, G.F.
6490
GILES, G.R.
5940*
GILGRANDE, L.A.
6061
GILL, A.M.
6014*
GILL, B.S.
6402*
GILLES, H.M.
6562
GILMAN, R.H.
6525
GINZBURG, S.N.
6446
GIOVINE, A.
5372*
GIRADELLO, D.
5515
GIROUX, L.
6487
GLOBL, H.J.
6172
GLOOR, F.
5704*
GLORIEUX, F.H.
6092*
GLUCK, F.W.
5900
GO, V.L.W.
5275*, 5286*, 5387*
GOBBEL, W.G., JR.
5297*
GODQUIN, B.
6592
GOEBELL, H.
5448, 6051*
GOETZE, H.
5583
GOHRBAND, G.
5785
GOIA, A.
6429
GOLBERG, L.
5477
GOLDEN, M.
5989
GOLDENBERG, H.
5418*
GOLDIN, V.A.
5759
GOLDMAN, P.
6019
GOLDSMITH, R.S.
5387*
GOLDSTEIN, H.M.
6484*
GOLDSTEIN, L.I.
5366*
GOLDSTEIN, N.P.
6344*
GOLIGHER, J.C.
6015*
GOMERCIC, H.
5606
GOMEZ, M.R.
6233
GONCHARENKO, E.N.
5578
GONZALES-BAYO, E.
6057*
GOOD, R.A.
5594*, 6091*
GOODMAN, D.S.
5355*
GOSH, T.E.
5710
GOTO, Y.
5324
GOTTESMAN, L.
6542
GOUFFRANT, J.M.
6152
GOUGH, K.R.
6580*
GOUT, J.P.
5870
GOUTIER, R.
5527
GOVONI, A.F.
5993
GRACE, D.M.
5891
GRACEY, M.
5566*
GRAHNEIS, H.
6531
GRAMBORT, D.E.
5400*
GRANDGIRARD, A.
5432
GRASBECK, R.
5213*
GRASEDYCK, K.
6119*
GRASSO, E.
6182, 6183
GRATADOUR, P.
5654
GREB, W.
5477
GREEN, R.D.
5272*
GREENBURG, A.G.
5310*

GREENE, L.J.
5352
GREENSTEIN, A.J.
6029*
GREIM, H.
6120*, 6124*
GRENIER, J.F.
5222, 5338*
GRETEN, H.
6123*
GRIEM, M.L.
5506
GRIFFEN, W.O., JR.
5399*
GRILL, W.
6409, 6449, 6451
GRIMBLE, R.F.
5456
GRIMELIUS, L.
5198*
GRIMMEL, K.
5422
GRINBERG, S.B.
6136
GRITSMAN, Y.Y.
5710
GROLLEAU, J.Y.
5972
GRONAUER, G.
6294*
GRUNEMEYER, R.
6462
GRONI, G.V.
5546
GROSFELD, J.L.
5570*
GROSS, R.
6292*
GROSSER, L.
5544
GROSSHANS, E.
6319
GROSSMAN, M.I.
5574*
GROSSMAN, M.L.
5271*
GROTE, W.
5554
GROTH, C.G.
6080*
GRUBB, M.N.
5267*
GRUBER, M.
5404*
GRUENAGEL, H.H.
6441
GRUN, M.
5397*, 6341*, 6343*
GRUNDMANN, E.
5736*
GRUTER, M.
6587
GUARINO, M.
5911
GUARNERI, L.
5315
GUAZZI, M.
6349*
GUDKOV, G.V.
5971
GUERAUD, J.P.
6122*
GUILLOU, PLJ.
5940*
GUIRALDES, E.
5241*
GUIVARC'H, M.
5945
GUJRAL, J.S.
5732
GULLIKSON, G.W.
5269*
GUPTA, P.P.
5473
GUPTA, S.
5992
GUPTA, V.J.
5239*
GUREVICH-RABKINA, F.P.
5837
GURLL, N.J.
5787
GURVICH, R.N.
5763
GUTH, P.H.
5597*
GUZMAN, I.J.
5894
GYR, K.
5694
HAAS, H.
6452
HAAS, K.
6219*
HABERLAND, G.L.
6065
HABESHAW, J.A.
5795
HADA, T.
6115*
HADCHOUEL, P.
5908*, 6448, 6453
HADHANYI, A.
5541
HADORN, B.
5214*, 5222, 5583
HADZIC, N.
6253
HAENSZEL, W.
5927*
HAFEZ, M.
6543
HAFF, R.C.
6075, 6469
HAFFEN, K.
5222
HAGE, G.
5304*
HAGEMANN, R.
5212*
HAGEMANN, R.F.
5234, 5581, 5589
HAGINO, Y.
5484
HAHNLOSER, P.
5878
HAKAMI, M.
5898
HAKANSON, R.
5206*, 5567*
HALGRIMSON, C.G.
6323
HALL, F.F.
6026*
HALLER, J.O.
6081*
HALLIDAY, J.W.
6114*
HALTER, F.
6558
HAMADA, F.
5295
HAMANI, A.
6167
HAMANO, K.
6040
HAMAYA, K.
6356
HAMILTON, S.R.
5723
HAMLYN, A.N.
6335*
HANDLER, S.D.
6219*
HANLEY, G.
6476*
HANSELL, J.R.
5903*
HANSEN, W.
6147
HANSKY, J.
6521
HANTSCHMANN, N.
5312*, 5327
HANY, Y.
6553
HANYO, F.
6040
HARADIN, A.R.
5783
HARANO, Y.
5629*
HARDING, J.D.
5200*
HARDY-SMITH, A.
5252*
HARGROVE, M.D., JR.
6571
HARMS, K.
5918
HARPER, P.S.
6485*
HARRIES, J.T.
5241*
HARRIS, P.J.
5360*
HARRIS, R.
6204
HARRIS, V.J.
6476*
HART, W.R.
6199
HARTEL, W.
5300*

HARTLEB, K.
5441
HARVEY, R.F.
5279
HARVEY, T.C.
6229
HASE, T.
5299*
HASEGAWA, J.
6011*
HASEGUWA, H.
6131
HASERT, V.
5674
HASHIMOTO, H.
6356
HASHIMOTO, N.
5595*
HASHIMOTO, Y.
5619*
HASHINOTSUME, M.
6115*
HASSAN, A.
6540
HASSAN, A.H.I.
6543
HASUMI, A.
6209
HATHOUT, S.
6540
HATTORI, N.
6131
HAUGER, W.
5679
HAUKI, H.P.
5222, 5583
HAUTEFEUILLE, P.
6028*
HAUTON, J.C.
5424, 5429
HAVA, M.
5268*
HAVEMANN, K.
6244*
HAY, J.M.
5577
HAYAKAWA, N.
5644
HAYAMA, T.
5319
HAYASHI, M.
6190
HAYASHI, Y.
5485
HAYMOND, M.W.
6197
HAYNES, R.C., JR.
5470
HAYS, D.M.
5392*, 5405*
HAYS, E.F.
5405*
HAYWARD, M.J.
5795
HEATLEY, R.V.
5600*
HECKERS, H.
6293*
HEDGES, J.R.
5245*
HEGARTY, M.M.
5855
HEGNER, D.
5353*
HEID, E.
6319
HEIKKINEN, E.
6002
HEINDENREICH, O.
5541
HEINE, H.
6133
HEINE, W.D.
6336*
HEINEMANN, R.
5458
HELD, H.
6150
HELLE, M.
6119*
HELMSTAEDT, D.
6228
HELMSTAEDT, E.R.
6406
HENDERSON, P.T.
5553
HENDERSON, R.D.
5718
HENGSTEBECK, W.
5675
HENK, R.
6148
HENN, R.M.
6593
HENNESSY, T.
6360
HENRICH, W.L.
6214*
HENRY, C.J.
5499
HENSLEY, G.
5506
HEPNER, G.W.
5403*, 5572*
HEPP, J.
6453
HERMAN, P.
5959
HERMS, G.
6252
HERNANDEZ, A.
5981
HERNANDEZ-GUIO, C.
6061
HERRERIAS, J.M.
W5664
HERRINGTON, J.L., JR.
5857
HERTZ, R.E.
6004
HERZ, J.E.
5390*
HESS, N.W.
6498
HESS, W.
5640, 6450
HEUTTINGER, M.
5418*
HIATT, J.
5599*
HICHINO, S.
5644
HIETANEN, E.
6404*
HIGASHINO, K.
6115*
HIGGINS, G.A., JR.
5933*
HIGGS, R.H.
6477*
HIKASA, Y.
5232
HILDREW, P.
6335*
HILL, M.J.
5928*
HIMAL, H.S.
5313, 5794, 5891
HINDFELT, B.
5462
HIRAMATSU, K.
6209
HIRAYAMA, H.
5295
HIRAYAMA, T.
5502
HIRCHE, H.
6245*
HIRSCH-MARIE, H.
5908*
HIRSCH, N.R.
6219*
HIRSCHHORN, N.
6486*
HIRSCHOWITZ, B.I.
5298*
HITCHCOCK, C.R.
6067
HO, K.J.
5536
HOBL, E.
6458
HOBSON, R.W., II
5585
HOFER, M.A.
5318
HOFFMAN, H.N., II
6090*
HOFFMAN, N.E.
5616*, 5620*
HOFFMANN, E.O.
5369*
HOFMANN, A.F.
5409*, 5616*, 5620*, 6102*
HOFMANN, J.W.
5286*
HOFSTAD, T.
5902*
HOGG, G.R.
6100*
HOHN, P.
5561*
HOKANO, M.
5228

HOLE, D.
5255
HOLLE, F.
5804
HOLLENDER, A.
5633*
HOLLENDER, L.F.
5815
HOLLENHORST, R.W.
6344*
HOLM, E.
5444
HOLMES, L.B.
6045*
HOLSING, N.
6518
HOLTZAPPLE, P.G.
5903*
HOLYCKE, E.D.
6032
HOMMA, T.
5229
HONDA, K.
5798
HONDA, R.
5322
HONDA, Y.
5482
HONEGGER, J.
5583
HONJO, I.
5380*, 5389*, 6046*, 6096*
HOPF, U.
5914, 6290*
HOPKEN, W.
6275
HOPPEL, C.L.
5525
HOREAU, J.M.
5972
HORII, Y.
5503
HORN, M.
6424
HORNICK, R.B.
6525
HORNSTEIN, G.P.
5905*
HOROVITZ, I.L.
6475
HORSLEY, S., III.
5936*
HOSHI, A.
5295
HOSHI, M.
5629*
HOSHITA, T.
5253
HOTCHKISS, J.L.
6313*
HOUGHTON, M.
5566*
HOUSSET, E.
5650
HOVASSE, P.
5876
HOVEY, L.M.
6000
HOWARD, E.W.
6000
HOWELL, K.
5374*
HOYUMPA, A.M., JR.
5247*
HRISTOZOVA, A.
5513
HSIEH, D.P.H.
5524
HUANG, S.N.
6223*
HUANG, S.S.
6579*
HUDSON, J.R.
5784
HUEHNERGARTH, R.J.
6214*
HUET, P.M.
6348*
HUGHES, J.H.
5979
HUGUET, C.
6167, 6448, 6581*
HUGUIER, M.
6171, 6581*
HULDT, G.
6550*
HULTON, L.
6523
HUMPHREY, E.W.
5933*
HUNTER, A.L.
5491
HUREAU, J.
5780
HURWITZ, A.
5268*
HUSBERG, B.
6323
HUTCHISON, G.A.
5298*
HUTTUNEN, R.
6002, 6198
HYASHI, M.
5502
IANISKER, G. IA.
6511
IBRAHIM, I.
5983
IDA, K.
5619*
IDA, T.
5389*, 6096*
IDE, G.
5757
IEDA, H.
5644
IKEDA, H.
5883
IKEDA, K.
5595*
IKINS, P.M.
5985
IMAI, H.
5622*
IMAI, M.
6040
IMAMURA, M.
5404*
IMPICCIATORE, M.
5289, 5328
IMPROTA, G.
5270*
IMRIE, C.W.
5854
INFANTE, R.
5430, 5480
INGHAM, P.A.
5251*
INNES, D.L.
5517
INOKUCHI, K.
5595*
IONOV, A.A.
6149
IORDACHESCU, D.
5475
ISAACS, R.A.
5563*
ISELER, G.
5407*
ISHCHENKO, V.T.
6156
ISHIDA, O.
5883
ISHIKAWA, M.
5622*, 6323
ISOBE, Y.
5643
ITIN, A.B.
6037
ITO, M.
5379*
ITO, T.
6072
ITOH, Z.
5322
ITOSHIMA, T.
5210*
IUSUBOV, D.R.
5813
IVANOVA, E.A.
5821
IVEY, K.J.
6521
IWAI, H.
5357*
IZMAILOV, G.A.
5873
JABLONSKA, M.
5809
JACEWICZ, M.
6537
JACK, D.
6552
JACKSON, F.L.
5892
JACKSON, K.L.
5509
JACOB, F.
5455
JACOB, R.H.
5603
JACOBS, G.
6407

JACOBSON, L. D.
5603
JACQUET, N.
5959
JAEGER, R.J.
5416*
JAGENBURG, R.
5867*
JAGENBURG, R.
5866*
JAILLARD, J.
6320
JAIN, S.
5732
JAKAB, F.
5961
JAKOWIDIS, T.
5841
JAKUBOWSKA, D.
6496
JAMES, A.E., JR.
5605, 5635*, 5934*
JAMES, D.G.
6106*
JAMES, I.M.
6335*
JAMES, J.H.
5467
JAMES, O.
6402*
JANEV, P.
6079*
JANKOWSKI, A.
6439
JANSEN, H.H.
5947
JANVRIN, S.B.
5789
JARRETT, I.G.
5522
JAULIN, P.
6255
JAYASEKARA, G.
5792
JEANMART, L.
6363
JEEJEEBHOY, K.N.
5408*
JELEN, J.
5776
JELEVA, M.
6527, 6532
JELLINEK, M.
5492
JENKINSON, S.G.
6571
JENNY, S.
5824
JENSEN, H.E.
5850
JERINA, D.M.
5530
JERUSALEM, C.R.
5447
JERVIS, H.R.
5205*
JESSE, R.
6369
JEYNES, B.J.
5202*
JEZEQUEL, A.M.
6194
JIMENEZ, G.
6304
JIMENEZ, J.
W5664
JINGU, K.
6083*
JOFFE, N.
5735*
JOHNSON, G.E.
5263
JOHNSON, L.R.
5285*, 5591*
JOHNSON, P.K.
6201
JOHNSON, W.D., JR.
6332
JOLLOW, D.J.
6218*
JONES, C.W.
5794
JONES, L.A.
5261
JOONG, S.T.
5502
JORDAN, P.H.
5725
JORDAN, P.H., JR.
5933*
JORGENSEN, B.
6116*
JOS, J.
5907*
JOSKE, R.A.
5401*
JOSSO, A.
5972
JOUBAUD, F.
6353
JOVANOVIC, D.A.
5508
JUNEMANN, A.
5964
JUSSILA, J.
6404*
KABOTH, V.
6277
KAKEMI, K.
5262
KALANT, H.
5367*, 5377*
KALASHNIKOV, S.A.
6494
KALINA, Z.
6016*
KALININ, A.V.
5827
KALLAI, L.
6253
KALLFELZ, F.A.
5293
KALSER, M.H.
5784
KAMEGAYA, K.
6209
KAMETAKA, M.
5468
KAMINSKI, D.L.
5381*, 5492
KAMINSKI, K.
6016*
KAMIYA, T.
5644
KAMME, C.
6578*
KANAI, T.
5295
KANATO, M.
5232
KANG, K.Y.
6115*
KANTO, J.
6467
KANWAR, Y.S.
5781
KAPLAN, E.
5614
KAPLAN, E.L.
5320, 5339*
KAPLAN, J.E.
5391*
KAPLAN, M.M.
6113*
KAPLAN, S.
5223
KAPPAS, A.
5356*
KAPRON, A.M.
5974
KARAMAN, N.V.
6436
KARDEL-MIZERSKA, T.
5776
KARDEL, T.
6337*
KAREVA, A.I.
5773
KARIPINENI, R.
5586
KARJALAINEN, P.
6404*
KAROLCEK, J.
6529
KARPENKO, L.N.
5336
KARPPANEN, H.
5302*
KARRAN, S.J.
5465
KARUZINA, I.I.
5451
KARVOUTZIS, G.G.
6235*
KASAHARA, T.
6190
KASKEVICH, L.M.
5821
KASSEM, A.
6158
KASSEM, A.S.
6543
KATEVUO, K.
6467

KATON, R.M.
5618*
KATSITADZE, T.K.
6281
KATSCHIS, C.
6570
KATUNUMA, N.
5354*, 5359*
KATZ, J.
5518
KATZ, S.
5931*
KAUFER, C.
6392*, 6396*
KAUFFMAN, G.L., JR.
5311*
KAUFFMANN-MACKH, G.
5286*
KAUFMANN, H.J.
6172
KAWAI, K.
5619*
KAWARADA, Y.
5459
KAZEM, I.
6085*
KAZIMIRCHIK, A.P.
5681
KEATING, J.P.
6197, 6473
KEDINGER, M.
5222
KEEFFE, E.
6346*
KEEHN, R.J.
5933*
KEIGHLEY, M.R.B.
6474
KEILER, A.
5958
KEISER, H.R.
6218*
KELLER, R.J.
5617*
KENDALL, F.M.
5517
KENNAUGH, R.C.
6225*
KERIBIN, D.
5972
KERLER, R.
5407*
KERSTEIN, M.D.
6009
KERSTEN, M.D.
6086*
KESTENS, P.J.
6364
KESTER, R.C.
5296*
KESWANI, R.K.
5988
KETYI, I.
6530
KETYL, I.
6528
KEUSCH, G.T.
6537
KHADEMI, M.
6066
KHANNA, J.M.
5377*
KHAZANOV, A.I.
5827
KHILNANI, M.T.
5617*
KHOCHAVA, A.I.
6283
KHUNDANOV, L.L.
6502
KIBRITE, A.
6382*
KICZKA, W.
6254
KIEFFER, R.
5726
KIENZLE, M.G.
5260
KILAM, S.K.
5892
KIM, S.J.
6564, 6565
KIM, Y.
5519
KIM, Y.S.
5519, 5563*
KIMURA, A.
6162
KIMURA, K.
5228
KINDLER, V.
6273
KING, J.E.
5929*
KING, P.C.
5910*
KINO, I.
5948
KINOSHITA, G.
5406*
KINOSHITA, N.
5316
KINTSCHER, M.
5441
KINZIE, J.J.
5506
KIREEVA, D.V.
5761
KIRKPATRICK, W.
5998
KISELEV, A.I.
6077
KISER, J.
6541
KISHI, S.
5645
KISILY, N.P.
5865*
KITA, S.
5231
KITAMURA, O.
6046*
KLAINER, A.S.
5930*
KLATSKIN, G.
6117*
KLAUSHOFER, K.
5292
KLEIN, H.J.
5778, 5779
KLEIN, W.
5477
KLEINTEICH, B.
6381*
KLEISBAUER, J.P.
5669
KLETZIEN, R.F.
5264
KLEY, H.K.
6127*
KLIMOV, V.N.
5816
KLINE, M.M.
5306*
KLOSE, G.
6123*
KLOSS, J.
6132
KLOTZ, D., JR.
6193
KNAUER, C.M.
6217*
KNEZEVIC, S.
6253
KNIGHT, D.H.
6056*
KNIGHT, W.B.
6569
KNOBLAUCH, M.
6042
KNOCKE, K.W.
6275
KNOLLE, J.
5914
KNOP, J.
5601, 5602
KNOWLES, B.R.
6114*
KNUTSON, C.D.
5697
KOBAYASHI, R.
5231
KOBAYASHI, S.
5658, 6040
KOCH, G.
5946
KOCH, H.
6400*, 6589
KOCH, M.
6194
KOCH, M.R.
5460
KOCHEN, M.
6338*
KOCIAN, V.J.
5916
KOCIANOVA, J.
5916
KOCK, H.J.
6384*
KOCK, N.G.
5866*, 5867*
KOGA, A.
5230

KOGA, Y.
5392*, 5405*, 5595*
KOHLER, P.H.
6237*
KOIDE, A.
5643
KOIKE, T.
5888
KOISCHWITZ, D.
5771
KOJIMA, K.
6052*
KOKAREVA, N.S.
5451
KOKOSCHKE, R.
5869
KOKOTEK, H.
6422
KOKUE, E.
5319
KOLAWOLE, T.M.
5986
KOLLMANN, D.
5918
KOLYADYUK, I.V.
5750
KOMARKOVA, O.
5760
KOMMERELL, B.
6274, 6300, 6378*
KONDO, T.
5644
KONDOVA, P.
6435
KONNO, T.
6162, 6536
KONNOVA, L.V.
5710
KONO, M.
5253
KONTUREK, S.
5331, 5332
KONTUREK, S.J.
5333
KOPPENFELS, R.
5715
KORDAC, V.
6094*
KORDES, B.
6387*
KORKUSHKO, O.V.
5330
KORMAN, M.G.
5409*, 5616*, 5620*, 6102*
KORN, P.
5869
KORNILOV, IU.M.
5775
KOROLENKO, I.A.
5436
KORPOVICH, G.A.
5609
KORTE, F.
5477
KORUCU, B.
6552
KOSAKA, K.
5231
KOSAREV, V.A.
6146
KOSILOVA, L.M.
6265
KOSS, N.
6009
KOSSAKOWSKI, R.
6250
KOTKO, D.N.
5330
KOTOVA, S.M.
5744
KOTTMEIER, P.K.
6193
KOVACHEV, P.
6532
KOVALEVA, N.B.
6140
KOYAMA, K.
5504
KOZHURA, V.L.
5427
KOZLOV, O.A.
5648
KOZLOV, V.I.
5326
KOZLOVZ, A.V.
5941
KOZUKA, T.
5883
KRADINOV, A.I.
5772
KRAEHENBUHL, J.R.
5636*
KRAJKA, C.
6434
KRALIK, J.
5730
KRAMANN, B.
6368
KRAMAR, R.
5418*
KRAMER, D.
6228
KRAMER, R.
6275
KRASILNIKOV, K.B.
5277*
KRASNOPROSHINA, L.I.
6510
KRASTEV, M.
5943
KRAUSE, W.D.
6441
KRAVCHUK, A.N.
6416
KREBS, H.A.
5412*
KREJS, G.J.
5852
KREMP, F.
6460
KRENTZ, K.
5785
KRETSCHMER, G.
5444
KRETSCHMER, K.P.
5995
KRICHEVSKII, IU. A.
6263
KRIEG, H.
6394*
KRIEGER, R.I.
5524
KRISTENSEN, M.
5812
KRITCHEVSKY, D.
5538
KRIVORUCHKO, B.I.
5382*
KROGER, W.
5674
KROKER, R.
5353*
KROPFF, G.
5972
KRUSKEMPER, H.L.
6127*
KUBEI, V.I.
6414
KUBICEK, V.
6060
KUDO, S.
6115*
KUDRIASHOV, IU. B.
5578
KUENZLE, C.C.
6213*
KUGLER, S.
5946, 6583
KUHLMANN, H.W.
5975
KUHN, H.H.
6585
KUHR, J.
5754
KUKSIS, A.
5457, 5493, 5540
KULIK, V.P.
5219
KUMAR, R.
6481*
KUMAR, V.
6517
KUME, M.
5226
KUMMERLE, F.
6585
KUNLIN, A.
5577
KUNO, Y.
5502
KUNTZ, J.L.
6038
KUNZ, B.
5674
KURAMOTO, T.
5253
KURBANGALEEV, S.M.
6444
KURDYBAILO, F.V.
6301
KURLOVA, V.I.
6511
KURODA, C.
5883

KURODA, Y.
5887
KURONO, C.
5504
KURTZ, R.
6516
KURTZ, R.G.
5979
KURUP, C.K.R.
5531
KUSHCH, N.L.
5217
KUSKA, J.
5434
KUWERT, E.
6245*
KUZIN, M.I.
5758, 5817
KUZIV, K.F.
6418
KUZMINA, N.A.
6058
KVEY, K.J.
5982
LA BARRE, J.
5842
LA COURANDERSON, J.
5768
LAAR, H.J.
6150
LACALIO, S.A.
5514
LACAZE, J.
5708
LACOSTE, A.M.
5472
LAFONT, H.
5429
LAFONT, J.
5199*
LAGER, I.
5867*
LAGI, A.
6354
LAIRON, D.
5429
LAKHTINA, V.P.
5326
LAKIN, R.C.
5415*, 5533
LAKSHMANAN, M.R.
5529
LAKY, D.
6153
LAKY, M.
6082*
LAM, A.Y.
6036
LAM, C.R.
5797
LAMABADUSURIYA, S.P.
5241*
LAMBERT, H.P.
6477*
LAMBERT, P.W.
5387*
LAMBILLIOTTE, J.P.
6364
LAMBOEUF, Y.
5254
LAMBRECHTS, A.
6493
LAMPRECHT, J.
5220
LANAS LARRANAGA, E.
6138
LANCASTER-SMITH, M.J.
6021
LANCASTRE, F.
6555
LANDER, H.
5634*
LANDRISCINA, C.
5545, 5546
LANDRY, M.
5638*
LANE, A.E.
5244*
LANE, M.D.
5471
LANGLEY, P.G.
6089*
LANGNESS, V.
6119*
LAPALUS, F.
5547
LAPLANTE, L.
5751
LARGIADER, F.
5824, 6042
LARMI, T.K.I.
6002, 6198
LAROUZE, B.
6248*
LARRIEU, A.J.
5726
LARSON, S.M.
5699
LARSSON, L.I.
5206*, 5567*
LARUSSO, N.F.
5616*, 5620*
LASAGNA, L.
5562*
LATENKO, Y.P.
6262
LATSINIK, G.E.
6264
LAUCHART, W.
5398*
LAURENCELLE, L.
6487
LAVAL, P.
5669
LAVALLARD, M.C.
6167
LAVERDANT, C.
6158, 6270, 6453
LAVERY, J.P.
5683
LAVIGNE, J.E.
6047*
LAVOIE, P.
6348*
LAVROVSKAIA, V.M.
6511
LAW, D.H., IV.
5737*
LAWRENCE, W., JR.
5936*
LAZARO, E.
6066
LAZERTE, G.D.
5995
LE FROCK, J.L.
5930*
LE MASSON, J.M.
6554
LE TALLEC, Y.
6584
LEA, M.A.
5460
LEACH, K.G.
5465
LEBENTHAL, E.
5924
LEBLANC, A.E.
5367*
LEBODIC, L.
5216*
LEBODIC, M.F.
5216*
LEBRANCHU, Y.
5870
LECHAGO, J.
5593*
LEDGER, W.J.
6534
LEE, F.H.
5453
LEE, S.S.
5536
LEE, Y.K.
5518
LEEVY, C.M.
6312*, 6325, 6328
LEFEVER, G.S.
5272*
LEFKIDIS, C.
5853
LEFRANC, G.
5216*, 5334
LEGGE, D.A.
6360
LEGORRIETA, J.
6525
LEHMANN, H.
6242*, 6278
LEHRNER, L.M.
5344*
LEINWEBER, B.
6293*
LELBACH, W.K.
5754, 6326, 6339*, 636
LENDRUM, R.
6055*
LENKEI, R.
6082*
LENNARD-JONES, J.E.
5258, 6013*
LENOIR, G.
5907*
LENRIOT, J.P.
5876

LEO, M.
6373
LEONHARDT, H.
6338*
LEONIDAS, J.C.
5623*
LEOPOLD, G.R.
5702
LERY, N.
6154
LESAGE, R.
6487
LESCHENKO, V.M.
5875
LESHER, J.
5212*
LESHER, S.
5212*, 5589
LESTER, D.
5361*
LETOUMEUR, F.
5880
LEUSCHNER, U.
6391*
LEUTSKYI, K.M.
5237*
LEVEDEV, S.P.
6258
LEVERETT, F.L.
5784
LEVIJ, I.S.
6240*
LEVIN, R.
5731
LEVITSKY, A.P.
5742, 6064
LEVORSTAD, K.
5719
LEVY, E.
6581*
LEWIN, M.
5225
LEWIS, E.A.
5986
LEWIS, G.P.
5317
LEWIS, J.E.
6217*
LEWIS, J.W., JR.
6009
LEWIS, M.L.
6238*
LEZHNEV, I.A.
6282
L'HERMITE, A.
5334
LIAPKOV, B.G.
5383*
LIASHKO, N.A.
5818
LICHTENBERGER, L.M.
5285*, 5591*, 5593*
LIEBER, C.S.
5487, 6327
LIEBERMAN, I.
5555
LIEBERMAN, J.
6333*
LIEBOW, C.
5342*
LIEHR, H.
5397*, 6341*, 6343*
LIN, J.S.
5701
LIN, T.M.
5308*
LINDENAUER, S.M.
5856
LINDER, M.M.
5300*
LINDNER, J.
6119*
LINDNER, W.
6267
LINDSAY, D.B.
5522
LINDSTROM, C.
5932*
LINHART, P.
6274
LINNEMAN, C.C., JR.
6093*
LINZELL, J.L.
5522
LISKER, R.
5920
LISOCHKIN, B.G.
5761
LITINSKAIA, E.V.
5821
LIVNI, N.
6240*
LJUNGSTROM, I.
6550*
LOCALIO, S.A.
5627*
LOEBENBERG, D.
6576
LOFFLER, A.
6392*
LOFLAND, H.B.
5370*
LOHMANN, R.
6018
LOISEL, W.
5432
LOMACHENKOV, V.D.
5770
LOMBARDI, B.
5340*
LONCAREVIC, F.
6132
LONDON, W.T.
6248*
LONG, F.C.
5931*
LONGNECKER, D.S.
5340*
LOOSE, L.D.
5250*
LOPEZ GALLART, J.M.
5886
LOPEZ HABIB, G.
5920
LORMANS, J.A.G.
6545*
LOTH, J.
5377*
LOTZE, J.
6228
LOVETT, W.L.
5936*
LOYGUE, J.
6581*
LUCEA, C.
5607
LUCHSHEV, VI.
6282
LUCKING, T.
5671
LUDWIG, J.
6090*
LUMERMAN, H.
6535
LUNDQUIST, F.
5489
LUNDY, J.
6516
LUNZER, M.R.
6223*
LUSZAWSKA, D.
5278
LUTZ, W.
6084*
LYMAN, R.L.
5341*
MACHIEDO, G.W.
6047*
MACIAS, A.
W5664
MACKAY, A.M.
6013*
MACKAY, I.R.
5466
MACKENZIE, R.J.
5384*
MACKIE, J.A.
5782
MACLEAN, L.D.
5794, 6347*
MADAYAG, M.A.
5627*
MADURA, J.A.
5570*
MAEDA, Y.
5887, 6356
MAEKAWA, T.
5883
MAFART, Y.
6555
MAFFIOLI, C.
6039
MAGALHAES, M.C.D.
5486
MAGNUSSON, A.
6479*
MAGNUSSON, B.
6479*
MAGOT, T.
5579
MAGRINI, F.
6349*

MAHAJAN, R.C.
5557, 6549*
MAHLER, D.
6346*
MAHMOOD, A.
5243*
MAIER, P.
6243*
MAINES, M.D.
5356*
MAIOROV, V.M.
5763
MAK, E.
5408*
MAKAROVA, L.A.
6489
MAKAROVA, L.M.
5219
MAKINO, K.
5228
MAKSIMENKOVA, A.N.
5219
MAKSIUTINA, N.P.
6379*
MALACCO, E.
6305
MALACINSKI, G.M.
5344*
MALAFOSSE, M.
6581*
MALHOTRA, Y.
6032
MALINOWSKI, J.
6431
MALLIA, A.K.
5355*
MALYUKH, L.S.
5828
MAMEDOV, I.M.
5966
MAMONTOV, A.S.
5710
MANALIGOD, J.R.
5781
MANGAN, J.L.
5522
MANHAL, J.
6060
MANN, N.S.
5695
MANOLOV, G.
5943
MANSBACH, C.M., II
5569*
MANSI, C.
5294
MANSOUR, E.G.
6206
MANTOVANI, A.
5515
MANUILENKO, B.A.
5950
MANZANOS, A.
5607
MARAI, L.
5457, 5493, 5540
MARANO, V.
6207
MARCHAND, M.
5876
MARCOULLIS, G.
5213*
MARECHAL, R.
5527
MARGOLIS, S.
6049*
MARI, S.
5948
MARINONE, C.
6355
MARKELOVA, V.F.
5383*
MARKIEWICZ, A.
5925
MARKOFF, N.
6458
MARKS, C.
5864*
MARMORSHTEIN, S.Y.
5740
MARQUAND, J.
5945
MARRYATT, G.
5718
MARSH, J.
5857
MARTIMBEAU, P.W.
5862*
MARTIN-DUPONT, M.C.
6503
MARTIN, J.S.
5517
MARTINEZ, F.
6525
MARTINEZ LOPEZ DE LETONA, J.
6170
MARTINI, G.A.
6173, 6505
MARUHAMA, Y.
6202
MARX, R.
5660
MASCIARIELLO, S.
5922
MASEVITCH, C.H.
5865*
MASSARRAT, S.
6387*
MASSE, G.
5727
MASTROFRANCESCO, B.
6024*
MASUDA, M.
5503
MASUGI, Y.
5501
MASURKA, V.
5949
MATEEV, V.
6435
MATERN, S.
5388*
MATIASHIN, I.M.
5845
MATOLO, N.M.
5861*
MATSUDA, T.
5643
MATSUMOTO, M.
5253
MATSUMURA, K.
5245*
MATSUNO, K.
6068
MATSUNO, T.
5644
MATSUZAKI, O.
5757
MATTHES, K.J.
6293*
MATTHEW, H.
6231
MATUCHANSKY, C.
5257, 6028*
MATUSZEWSKI, W.
6016*
MATVEEV, N.K.
5763
MAUERBERGER, H.
6294*
MAUPAS, P.
6248*
MAY, B.
6228
MAY, C.
5373*
MAYER, C.J.
5274*
MAYER, G.
6184
MAYER, S.
6184
MAYRON, L.W.
5614
MAYS, E.T.
6129*, 6205
MAZURIN, A.V.
6488
MAZZACCA, G.
5922
MAZZETTI, M.
6456
MCCAFFERY, T.D.
6546*
MCCALL, J.T.
6344*
MCCALLUM, R.W.
5306*
MCCANN, V.J.
6097*
MCCREADIE, S.R.
6110*
MCDONAGH, A.F.
5461
MCDONALD, I.R.
5611
MCDOUGALL, R.M.
6376*
MCFARLAND, J.
5802*
MCHENRY, L.C., JR.
6345*
MCINTIRE, K.R.
6117*

MCINTYRE, N.
6361
MCKEOWN, M.D.
5859
MCKINLEY-MCKEE, J.S.
5528
MCLAUGHLIN, M.H.
5310*
MCLELLAN, J.S.
6229
MCLEOD, J.W.
5392*
MCMAHON, P.J.
5989
MCMILLAN, W.B.
5783
MCMULLIN, J.P.
5989
MCSHERRY, C.K.
5386*
MCVIE, J.G.
5795
MEEROFF, J.C.
5275*, 5738*
MEGAHED, G.M.
6003
MEGLI, F.M.
5545
MEHLMAN, B.
5299*
MEINGARD, F.
6252
MEKHANIK, Z.I.
6301
MELCHIORRI, P.
5270*
MELERO CALLEJO, E.
6061
MELLIN, V.M.
5747
MELLO, M.L.S.
5211*
MELLO, P.
6217*
MELLON, P.J.
6089*
MELNYK, C.
6346*
MELNYK, C.S.
6214*
MELON, G.
5959
MELOTTI, G.
6459
MENSHIKOV, V.V.
5758
MERADJI, M.
5722
MERINO ANGULO, J.
6170
MERKEL, H.
5446
MERTZ, D.P.
5649, 6340*
MERZLIAK, M.N.
5578
MESHALOVA, A.N.
6511
MESNIER, J.M.
5976
MESSINA, L.
6121*
MESSINETTI, S.
6001
MESSINI, M.
6307
METADENOV, V.
6438
METAIS, P.
5363*
METCALFE-GIBSON, C.
5793
MEVES, M.
5823
MEYER-BURG, J.
5628*
MEYER ZUM BUSCHENFELDE, K.H.
5914, 6276, 6290*
MEYNIEL, M.G.
5547
MICHAS, C.A.
5860*, 5861*
MICHEL, H.
6366
MICHEL, R.
5426
MIDDLETON, H.M.
5247*
MIDHA, D.
6389*
MIEDERER, S.E.
6392*
MIELI, C.
5667
MIGNECO, G.
6306
MIHAS, A.A.
6314*
MIKA, H.
5561*
MIKKELSEN, K.
5314
MILANESE, M.
5642
MILKOVIC, S.
5560
MILLEN, B.H.
5592*
MILLER, B.
6505
MILLER, F.J., JR.
5635*
MILLER, M.L.
5223
MILLETTE, B.
6348*
MILLIGAN, F.D.
5897
MILLMAN, I.
6248*
MILMAN, M. SH.
6268
MILNE, R.W.
5604
MINICONI, P.
6375
MIRANDA BAIOCCHI, R.
6061
MIRONOVA, K.A.
5818
MIROSHNICHENKO, V.A.
6488
MISEK, J.
5949
MISIEWICZ, J.J.
5800*
MISRA, A.K.
5926
MISRA, U.K.
5549
MISSJURA, N.V.
5774
MITAL, I.
5534
MITAL, R.V.
6010
MITCHELL, A.B.S.
6014*
MITCHELL, J.R.
6218*
MITIS, M.
5332
MITKOV, D.
5513
MIURA, Y.
5357*
MIYAZAKI, H.
5502
MIYOSHI, A.
5887
MIZEJEWSKI, G.J.
5417*
MIZUNO, H.
6069
MOCHMANN, H.
6533
MOCKEL, G.
6385*
MODIGLIANI, R.
5257, 6028*
MODY, B.
5857
MOHR, P.
6042
MOINE, D.
6453
MOKKA, R.
6198
MOLCHANOV, A.I.
6372
MOLDENHAUER, W.
5674, 5678
MOLINIE, C.
6270
MOLINO, G.
5642, 6355
MOLNAR, J.
6443
MONETTI, G.
5335
MONTALI, R.J.
5934*
MONTET, A.M.
5424

MONTET, J.C.
5424
MONTPELLIER, J.
5216*
MOODY, F.G.
5207*, 5590*
MOOKERJEA, S.
5493, 5540
MOONEY, C.S.
5399*
MOONEY, R.A.
5471
MOORE, G.
6112*
MOORTHY, B.
6517
MORA, M.A.
5920
MORDVINKINA, T.N.
5840
MOREAU, M.F.
5547
MORENO, M.
W5664
MOREV, A.G.
6372
MORI, S.
5883
MORICI, V.
5755
MORIMATSU, M.
5887
MORIN, A.
5972
MORISOT, P.
6167
MORL, M.
6587, 6588
MORUSINI, S.
6362
MOROVATI, S.S.
5981
MORRIS, H.P.
5460
MORRIS, J.S.
5632*
MORRIS, T.J.
5255
MORRISSEY, K.P.
5386*
MORTON, M.S.
5255
MORTON, W.J.
5728
MOSAVY, S.H.
5898
MOSCATO, V.
5753
MOSCHINSKI, D.
6407
MOSIN, V.I.
5960
MOSLEHY, A.
5898
MOSLEY, J.W.
6235*
MOSSELMANS, R.
5375*
MOSSINI, F.
5328
MOST, H.
6573
MOSTBECK, A.
5637*
MOTA, G.
6082*
MOTAI, Y.
5887
MOTTA, G.
6495
MOTTA, P.
5215*
MOTTON, G.
5713, 5714
MOTUZNYI, V.A.
5276*
MOUCHET, A.
5945
MOULT, P.J.A.
6230
MOURRUT, B.
6366
MOWAT, A.P.
6208
MUDGE, G.H.
5362*
MUERKER-ALZER, G.
6292*
MUKHIN, A.S.
6258
MUKOJIMA, T.
6131
MULDER, H.
5999
MULDER, J.L.
5633*
MULE, A.
5428
MULHERIN, J.L., JR.
5857
MULLER-BERGHAUS, G.
5446
MULLER-BRAND, J.
5673
MULLER, G.
6211*
MULLER-KLUGE, M.
6185
MULLER, O.
5542
MULLER, R.
6275
MULLER, R.P.
5754
MULLER-REINHARDT, B.
6051*
MULLER-SCHOOP, J.W.
5594*
MUNDAY, K.A.
5360*
MURADKASYMOV, I.R.
6454
MURAKAMI, K.
5619*
MURAKAMI, T.
5210*
MURANISHI, S.
5262
MURATA, F.
5229
MURLIN, A.M.
6518
MURO, J.
6062
MURPHY, S.D.
5416*
MURR, H.
5660
MUTING, D.
6272
MUTO, H.
6040
MYHER, J.J.
5457, 5493, 5540
MYREN, J.
5693
NABESHIMA, Y.
5469
NADAI, T.
5226
NAEF, A.P.
5705*
NAGAO, K.
5757
NAGASHIMA, H.
5481
NAGASUE, N.
5512
NAGATA, A.
5229
NAGATA, T.
5229
NAGAYO, T.
5788
NAIR, C.R.
5376*
NAJARIAN, J.S.
5537
NAKAE, Y.
5644
NAKAJIMA, S.
6027*
NAKAMURA, H.
5883
NAKAMURA, K.
5615*
NAKAMURA, K.T.
6490
NAKAMURA, T.
5319
NAKAMURA, Y.
5615*
NAKANISHI, S.
5406*
NAKASHIMA, M.
5484
NALIN, D.R.
6478*
NAMBARA, T.
5459
NAPALKOV, P.N.
5846, 6077
NARASAKA, T.
5622*

NARDI, G.L.
6045*
NASCIMENTO, J.S.
6490
NASH, J.F.
5308*
NATHANSON, M.
6145
NAUER, E.
5663, 5712
NAVA, G.
5667
NAYAK, N.C.
5534, 5535, 6246*
NAYUDU, P.R.V.
5565*
NEAL, R.A.
5491
NECHAEVA, T.A.
6261
NECHAI, A.I.
5971
NECHIPORUK, V.M.
5847
NEGINA, IU. P.
6511
NEGKEL, R.
5568*
NELSON, R.S.
6484*
NELSON, T.G.
6000
NEPOKROEFF, C.M.
5529
NEPORENT, M.I.
6059
NERVI, F.O.
5358*
NESHAT, A.A.
6390*
NESS, G.C.
5529
NEUBURGER, P.
5976
NEUFELD, L.
5453
NEUGODOV, V.A.
5763
NEUMANN, F.
5769
NEUMAYR, A.
5637*
NEVALAINEN, T.J.
5208*
NEVILLE, E.
6106*
NEVZIL, E.
5472
NEWCOMER, A.D.
5863*
NEWMAN, A.
5800*
NGUYEN-VAN-HANH
5890
NICOLINI, C.
5385*
NICOLO, G.
5915
NICOLSON, M.C.
5392*
NICULESCU, S.
5475
NIELSEN, O.V.
6116*
NIEMAN, W.H.
5386*
NIESCHLAG, E.
6127*
NIESSEN, K.H.
5919
NIIZUMA, S.
5658
NIKIFOROV, V.N.
6264
NIKITIN, G.A.
5803, 5830
NIKOLAEVA, V.V.
6296*
NIKOLAIEV, A.V.
6146
NILSSON, G.
5567*
NIMURA, Y.
5044
NIRANKARI, O.P.
5376*
NISEVICH, N.I.
6261
NISHI, K.
5280
NISHIMURA, M.
5230
NISHIMURA, T.
6190
NISTICO, G.
6207
NIVATVONGS, S.
6006
NIZZA, P.
5577
NOACK, R.
6384*
NOBLE, A.R.
5360*
NODA, A.
6027*
NOETZEL, H.
6370
NOGUEIRA, C.E.D.
6053*
NORBERG, H.P.
5339*
NORDLINGER, B.
6167
NORMAN, M.E.
5903*
NORTON, L.
6112*
NOTO, N.
6068
NOTTEN, W.R.F.
5553
NOVO PREGO, C.
6170
NOVY, S.
5998
NOZICKA, Z.
5760
NUBILE, G.
6181
NUESCH, H.J.
5824
NUMAZAWA, M.
5622*
NYMAN, K.E.
5929*
OATES, G.D.
6474
OBERLING, F.
6184
O'BRIEN, W.
6548*
OBWALD, V.H.
5541
O'CALLAGHAN, S.E.
5724
OCHOA, P.
5563*
OCKLITZ, H.W.
6533
O'CONNELL, A.
6245*
ODA, M.
5229, 5368*, 5408*
ODERDA, G.
5912
OEHLER, G.
6148
OEHLERT, W.
5209*, 6243*
OGANESIAN, O.A.
6308
OGATA, H.
5262
OGATA, K.
5469
OGAWA, N.
5485
OGBORN, A.D.R.
5906*
O'GRADY, J.F.
5989
OH, S.Y.
5552
OHASHI, Y.
5231
OHBA, H.
5501
OHI, I.
6040
OHISAKA, K.
5228
OHKOCHI, T.
6115*
OHKURA, H.
6131
OHLENSCHLAGER, G.
6186
OHNEDA, A.
6202
OHNEMUS, H.
5448
OHNISHI, H.
5485

OHNO, T.
6131
OHNUMA, I.
5843
OHTANI, R.
6115*
OHTSUKA, H.
5357*
OHTSUKI, M.
6202
OKABE, S.
5798
OKANGUE, T.
5503
OKAZAKI, N.
6131
O'KEANE, M.A.
5384*
OKHONKO, V.I.
5818
OKHRIMOVICH, L.M.
5820
OKSMAN, F.
6584
OKUDA, K.
5238*
OKULOV, V.B.
6149
OLBERMANN, M.
5914, 6063
OLDKEY, T.B.N.
5801*
OLEKSON, V.I.
6418
OLEKSY, J.
5834
OLINESCU, R.
5464, 5476
OLIVA, J.
6304
OLIVAKI, M.T.
6349*
OLSEN, W.
5246*
OLSEN, W.A.
5269*
OLSEN, W.R.
6203
OLSSON, R.
6525
OMAR, M.
5930*
OOTA, K.
5948
OPFERKUCH, W.
6290*
OPOLON, P.
6167
ORATZ, M.
5488
ORCHARD, R.T.
6014*
O'REILLY, K.
6200
ORLANDI, F.
6194
ORLOFF, M.J.
5400*, 6322
ORLOWSKA, J.
5913
ORTEGA VEGA, J.
6138
ORTIZ VAZQUEZ, J.
6062
OSADCHY, B.D.
5742
OSAKA, A.
5510
OSBORNE, J.W.
5233, 5587, 5588
OSMAN, A.IU.
5345
OSSWALD, P.
6211*
OSTEAUX, M.
6363
OSTER, J.R.
6313*
OSTER, Z.H.
5699
OSTROVSKAIA, M.S.
5971
O'SULLIVAN, P.
5987
OTTENI, F.
5815
OTTO, G.
6221*
OTTO, K.
5955
OTTO, P.
5956
OTTOLENGHI, G.
5669
OUSHOORN, H.H.
5921
OUSTRIERES, G.
6555
OVCHINNIKOV, V.I.
5765
OVERHOLT, B.F.
5698
OWEN, C.A., JR.
5394*
OYESIKU, J.E.J.
5241*
OZAWA, K.
5380*, 5389*, 6046*, 6096*
OZEGOVIC, B.
5560
OZZELLO, L.
5705*, 5969
PADIEU, P.
5449
PAGE, D.L.
5737*
PAIDLICK, A.
5447
PAIKOV, V.L.
5874
PAINTER, T.D.
5783
PALADE, G.
5351
PALADE, G.E.
5374*
PALASCIANO, G.
5287*, 5304*, 5337*, 5348
PALIARD, P.
6154
PALMA, F.D.
5714
PALME, G.
6338*
PALMER, W.K.
5495
PANCHENKO, K.P.
5745
PANCHISHINA, M.V.
5743
PANINI, S.R.
5531
PANITCH, N.
6593
PANNACCIULLI, I.
5294
PANOUSE-PERRIN, J.
6279
PAPAVASSILIOU, K.
5649
PAPENBERG, J.
5414*
PAPLIYAN, N.P.
5710
PAQUET, K.J.
6342*
PARAF, A.
6350
PARAMIO RUIBAL, A.
6160
PARC, R.
5577
PARDO REDONDO, A.
5917
PAREDES, S.
5987
PARENT, J.A.
5618*
PARIS, J.
6287, 632
PARIS, J.C.
6320
PARIZA, M.W.
5264
PARKIN, J.V.
6522
PARKINS, R.A.
6014*
PARKS, J.S.
5903*
PARONI, R.
5608
PARTIN, J.C.
6093*
PAS, E.
5962
PASCAL, G.
5450
PASCU, E.
5476
PASQUIE, M.
5953
PASS, B.R.
5995

PASSARO, E., JR.
5593*, 5599*, 5734*, 6483*
PASTICIER, A.
6143
PATEK, A.J., JR.
6318*
PATTI, J.
6405*
PAUL, F.
5769
PAUL, H.
5263
PAULINI, K.
5422
PAUMGARTNER, G.
6118*, 6433
PAVELKA, M.
5292
PAWLAK, J.
6161
PAYET, M.
6143, 6248*
PAYNE, J.E.
6247*
PEABODY, J.
5731
PEARSE, A.G.E.
5198*
PEDERSEN, L.
6388*
PEDERSON, L.
5371*
PEDRINIS, E.
6558
PEITZMAN, S.
6071
PELERAN, J.C.
5543
PELLEGRINI, S.
5515
PELLET, B.
5969
PELOT, D.
5994
PENASSE, W.
5373*
PENNER, E.
5869
PENNEY, D.P.
5562*
PENTIKAINEN, L.A.
6317*
PENTIKAINEN, P.J.
6317*
PENTTILA, A.
5197*, 5576*
PEPPERCORN, M.A.
6019
PERACCHIA, A.
6362
PERBELLINI, A.
5713
PERCUDANI, M.
5608
PERDOMO, J.
5563*
PEREIRA, J.L.
6490
PEREY, D.Y.E.
5604
PEREZ, D.A.S.
6157
PEREZ JIMENEZ, F.
6062
PEREZ MIJARES, R.
W5664
PERGOLA, F.
5843
PERISSAT, J.
6152
PERISSEL, B.
5449
PERMIAKOV, V.N.
5816
PERNOW, B.
5567*
PESCHL, L.
5637*
PESKIN, G.W.
5310*
PESSAYRE, D.
6028*
PETEL, B.
5953
PETER, P.
5852
PETERS, P.E.
5639*
PETERS, R.L.
6249*
PETIT, D.
5430
PETITE, J.P.
5650
PETRI, M.
6116*
PETROVA, A.N.
5829
PETRUNIN, Y.P.
6260, 6261
PETTERSSON, C.
5633*
PETZOLD, H.
6428
PEZZOLI, C.
5548
PFANDER, W.H.
5526
PFEFFER, H.
6220*
PHAN, T.
5363*
PHILIPPAKOS, D.
5800*
PHILIPSON, B.
5866*, 5867*
PHILLIP, J.
6508
PHILLIPS, M.J.
5368*, 5408*
PHILLIPS, S.F.
5275*
PHILLIPS, T.M.
6560
PHLIPPEN, R.
6163
PICACHO, R.
6323
PICHLER, E.
6253
PIERON, R.
6555
PIETRI, H.
5669
PIHLAJAMAKI, K.
6467
PILON, R.
5199*
PINKERTON, J.A., JR.
5297*
PIOMBO, I.
6309
PIPER, P.J.
5317
PIRK, F.
5652
PIROGOV, A.I.
5710
PIRWITZ, B.
6216*
PISAREV, A.G.
6259, 6261
PITOL, A.
5920
PIYASENA, K.H.B.
6106*
PLAKSINA, G.B.
6371
PLATONOVA, O.N.
5970
PLAZA, J.
6519
PLAZZI, V.
5328
PLISKIN, M.E.
5511
PLOTNIKOV, IU. V.
5846
PLOW, E.F.
5688
PLUDRA, J.E.
6399*
POCHACZEVSKY, R.
5623*
PODDA, M.
6135
PODZIMEK, A.
6060
POGANY, CS.
5962
POGODINA, A.N.
6419
POHL, W.
5755
POIRIER, R.
5669
POKORA, J.
5739*
POKROVSKII, V.I.
6513
POLAK, J.M.
5198*
POLESE, A.
6349*

POLETTI, J.B.
5977
POLEY, J.R.
5624*
POLTERAUER, P.
6109*
PONGPIPAT, D.
6236*
PUPOV, P.
5826
POPOVA, T.V.
5941
POPOWSKI, A.
5373*
PORTER, G.A.
6346*
PORTER, J.W.
5529
POSEVITZ, L.
6542
POSTOLOV, P.M.
5221, 5749, 5774, 5817
POTASHOV, L.V.
6500
POTEKAEVA, M.A.
6256
POTET, F.
5225
POTTEAU, B.
5433
POTTER, V.R.
5264
POTTER, W.Z.
6218*
POUPON, R.
5431
POURZAND, A.
5731
POWELL, L.W.
6114*, 6529
PRADAL, G.
5216*, 5334
PRADHAN, D.J.
5985
PRAKASH, A.
6010
PRAT, Y.J.
6020
PRATT, J.H.
5862*
PRAZAK, J.
5809
PREHN, R.T.
5511
PREISEG, R.
5420
PREISIG, R.
6118*, 6210*
PREISS, D.U.
5309*
PREUSS, H.J.
5674
PREZIOSI, P.
6207
PRIEST, R.J.
5797
PRIKHODCHENKO, V.V.
5217
PRIKUPETS, V.L.
6417
PRINCE, D.L.
5990
PROSS, E.
6063
PUCHER, A.
5333
PUGLEEVA, V.P.
5966
PUKHKAYA, V.K.
5765
PUSCH, H.J.
6187
PUTNAM, C.W.
6080*, 6323
PUTTLER, D.L.
6534
PUURUNEN, J.
5302*
QUAGLIARIELLO, E.
5545, 5546
QUATTROCCHI, G.
6182, 6183
QUELOZ, J.
5656
QUINTON, A.
5665, 5654
QUORAISHI, A.H.
6474
RABES, H.M.
5407*
RADECKI, T.
5332, 5333
RAEDSCH, R.
6378*, 6386*
RAGNI, R.
6295*
RAGNOTTI, G.
5476
RAIH, T.J.
5286*
RAISSONIER, A.
5430
RAIZMAN, A.
5480
RAKOW, L.
5422
RALL, M.B.
6444
RAM, M.D.
6389*
RAMAKRISHNAN, S.
5474
RAMAKRISHNARAO, P.
5474
RAMALINGASWAMI, V.
5473, 6246*
RAMASSO, M.
5915
RAMBAUD, J.C.
5257, 6028*
RAMBAUD, P.
5871
RAMSBY, G.R.
5393*
RANA, S.V.S.
5516
RANDALL, C.L.
5361*
RANDALL, H.T.
6024*
RANEK, L.
6078*
RANKIN, J.G.
6331
RANSON, J.H.C.
5514, 5627*
RAPANT, V.
5730
RAPAPORT, F.T.
5514
RASA, G.
5753
RASENACK, U.
5397*
RASKIN, A.M.
5761
RASNITSINA, I.E.
6259
RASZKE, K.
5836
RATAJCZAK-MAJEWSKA, C.
5913
RATNER, J.T.
6468
RAUSS, K.
6528, 6530
RAVENTOS, A.
5630*
RAYNAUD, A.
6366
READ, A.E.
5923
REALINI, S.
5656
REBAGLIATI, M.
5965
REDDY, J.K.
6095*
REDEKER, A.G.
6235*
REDMAN, C.M.
5374*
REED, W.D.
5471
REEDYK, L.
6111*
REESE, R.S.
5590*
REGAN, J.F.
5977
REGELSON, W.
5936*
REGENT, D.
5677
REGULA, M.
6378*
REHFELD, J.F.
5307*
REHNER, M.
5889
REIDENBERG, M.M.
6330
REIFFERSCHEID, M.
5968, 6590

REIMANN, W.
6271
REINHARDT, B.K.
5821
REIS, H.E.
6245*
REISMAN, T.N.
5784
REITH, H.I.
5444
RELIMAN, M.
6519
REMIGIO, P.S.
6535
REMY, J.
5577
RENAULT, H.
5908*
RENGER, F.
6271
RENIS, M.
5372*
RESHETNIAK, T.V.
6513
RETTENBACHER-TAUBNER, H.
5637*
REUL, G.J., JR.
6464
REUTER, G.
6319, 6384*
REY, J.
5907*
REYNIER, M.O.
5423
REYNOLDS, C.H.
5528
REYNOLDS, E.O.
5416*
REYNOLDS, T.B.
6249*, 6551*
REZNIK, N.O.
5682
RHAHALI, A.
5665
RHEE, Y.S.
6567
RHODES, B.A.
5605
RHODES, J.
5255
RIBEIRO, J.E.F.
6053*
RIBEIRO, T.
6053*
RICCARDI, V.M.
6045*
RICCI, C.
5734*
RICCI, G.
6251, 6286
RICHARDS, V.
5896
RICHAUME, B.
5677
RICHENS, E.R.
6580*
RICHTER, K.
5674, 5678
RIEDE, U.N.
5218
RIESNER, K.
6073
RIGAT, A.M.
5582
RIMBAUT, M.C.
6189
RINETTI, M.
6175
RIOS-DALENZ, J.
5984
RITCHIE, J.K.
6013*
RITCHIE, JA.A
6005
RITCHIE, W.P., JR.
5265
RITLAND, S.
6126*
RITTER, U.
6426
RIUS, J.M.
6556
RIVA, A.
6495
RIVA, M.
5315
RIVERA-CALIMLIM, L.
5562*
RIZHANOVSKAIA, V.M.
5847
ROBAUSCH, T.
5218
ROBBERECHT, P.
5349
ROBERTS, D.M.
5801*
ROBERTS, I.M.
5466
ROBINSON, V.
5859
ROCHA, E.
6538
ROCHA E SILVA, M.
5564*
RODRIGUEZ, R.
6525
RODUNER, J.
5583
ROENSPIES, U.
5938*
ROESCH, J.
5415*
ROETHLISBERGER, B.
5705*
ROGALSKY, V.Y.
5935*
ROGERS, L.F.
5998
ROHNER, H.G.
5754
ROJO-ORTEGA, J.M.
5204*
ROKHLIN, E.A.
5765
ROLLIN, P.
5443
ROMANETS, V.A.
5681
RONAI, P.M.
5396*, 5634*
RONCORONI, L.
6362
RONDIO, H.
5653, 5811
ROSCH, J.
5533, 5689, 6214*, 6346*
ROSCH, W.
5631*, 6035, 6048*
ROSCHLAU, G.
6168
ROSE, R.C.
5572*
ROSEAU, E.
5974
ROSENBERG, G.M.
6468
ROSENBERG, P.
6226
ROSENGREN, J.-E.
5932*
ROSENSWEIG, N.S.
6526
ROSSELO, R.
5669
ROSSETTI, M.
5885
ROSSIGNOL, A.M.
5870
ROSWIT, B.
5933*
ROTERMUND, H.M.
6243*
ROTH, R.
5571*
ROTHER, M.
6244*
ROTHMAN, S.S.
5342*
ROTHSCHILD, M.A.
5488
ROTTHAUWE, H.W.
5909*
ROUCAYROL, J.C.
6350
ROUSSEAU, B.
5973
ROUT, W.R.
5205*
ROUTER, M.
5323
ROVERE, G.Q.D.
5713, 5714
ROWLAND, H.A.K.
5691
ROWLEY, D.
5601, 5602
ROY, J.P.
6382*
ROY, R.M.
5497
ROY, S.
6246*
ROZANOV, Y.L.
5740

ROZE, C.
5347
RUBAN, V.M.
5844
RUBAY, J.
5777
RUBENSTEIN, J.A.
5539
RUBIN, C.E.
5245*
RUBIO, P.A.
6464
RUDERT, W.A.
5555
RUFFNER, B.W.
5936*
RUIJS, J.H.J.
5662
RUIZ, R.
5980
RUMACK, B.H.
6231
RUOFF, H.J.
5301*
RUPPIN, H.
5905*
RUSH, B.F., JR.
6047*
RUWART, M.
5381*
RUWART, M.J.
5492
RYBAKOV, A.I.
5944
RYDER, K.W., JR.
5391*
RYLEY, J.F.
6561
RZHANOVICH, A.P.
6139
SAAKYAN, A.G.
6489
SABA, T.M.
5391*, 6125*
SACHDEVA, R.
6246*
SACK, D.A.
6486*
SACK, R.B.
6486*
SAEGESSER, F.
5938*, 5965, 5969
SAFWAT, Y.
6540
SAHEKI, T.
5354*, 5359*
SAIK, R.P.
5310*
SAILER, R.
5964, 6407
SAIMOT, G.
6143, 6245*
SAITOH, Y.
6068
SAKAMOTO, K.
6190
SAKHIBOV, Y.D.
5759
SALEN, G.
6128*
SALERNO, M.T.
6373
SALHAB, A.S.
5524
SALISBURY, J.G.
6345*
SALMON, P.A.
6111*
SALVI, D.P.
5848
SALVITSKI, B.N.
5827
SALZER, M.
6133
SAMOKHVALOV, V.I.
6494
SAMSOEU, M.
6319
SAMSON, E.
6382*
SAMSON, E.I.
5828
SANATGER, R.
5707
SANDOR, G.
6581*
SANO, M.
5232
SANTAMARIA, J.N.
6331
SANTILLANA LOPEZ, T.
5917
SANTINI, B.
5912
SANY, C.
6366
SARKANY, I.
6230
SARLES, H.
5287*, 5304*, 5337*, 5348, 6050*
SARNITSKII, I.P.
6156
SASSE, D.
5218
SATEGNA, S.
6041
SATO, K.
5888
SATO, M.
5883
SATO, T.
5644, 6068
SATTLER, E.L.
5446
SAUBERLI, H.
5824
SAUNDERS, D.R.
5245*
SAUNDERS, M.G.
6318*
SAVARINO, V.
5295
SAVARY, M.
5705*, 5709
SAVELIEVA, L.A.
6296*
SAVELJEV, V.S.
5775
SAVINKIN, Y.N.
5740
SAVINOV, I.P.
6447
SAVITSKY, I.V.
5609
SAVVAKIS, C.
6404*
SAWYERS, J.L.
5851, 5857
SCARPELLI, F.
5608
SCARPULLA, P.
5805
SCATLIFF, J.H.
5621*
SCHABOWSKI, J.
5739*
SCHACHT, U.
5964
SCHAEDLER, R.W.
6022
SCHAFER, P.K.
5771
SCHAFER, V.A.
5561*
SCHAFFNER, O.
6400*
SCHANG, J.C.
5222
SCHEIFFARTH, F.
6299
SCHENKEN, L.L.
5589
SCHENKER, S.
5247*
SCHICKEDANZ, H.
6381*
SCHINDLER, D.
6586
SCHIRMEISTER, J.
6461
SCHIRMER, G.
5771
SCHJONSBY, H.
5902*
SCHLAAK, M.
6242*, 6278
SCHLEISSNER, L.A.
6087*
SCHMARSOW, R.
5639*
SCHMID, M.
6042
SCHMIDT, B.J.
6490
SCHMIDT, F.W.
5769, 6228
SCHMIDT, G.
5674
SCHMIDT, K.
5919
SCHMIDT, L.
5423

SCHMITT, J.C.
6552
SCHMOLD, A.
5554
SCHMUTZER, K.J.
5977
SCHNEEMAN, B.O.
5341*
SCHNEID, M.
6274
SCHNEIDER, K.W.
6369
SCHNEIDER, R.
5214*
SCHOBER, A.
6276
SCHOBER, H.
6242*
SCHOENBORN, R.
5278
SCHOENE, B.
6150
SCHOENFIELD, L.J.
5366*
SCHOLTYSEEK, E.
5235
SCHOLZ, H.
6291*
SCHONBORN, H.
6063
SCHOPPE, W.D.
6273
SCHRAMM, W.
5660
SCHREIBER, S.
5486
SCHRIEFERS, K.H.
5881
SCHROTER, D.
6017
SCHROTER, G.
6323
SCHUBERT, H.J.
5778, 5779
SCHUBERT, S.
5544
SCHUBERT, W.K.
6093*
SCHUCK, M.
6060
SCHULAK, J.A.
5320
SCHULTE, G.A.
6452
SCHULZ, H.G.
6030*
SCHULZE, P.J.
5410*
SCHUMACHER, K.
6292*
SCHUMPELICK, V.
5288*
SCHUR, P.H.
6025*
SCHWANDT, P.
6134
SCHWARZ, H.P.
6118*
SCHWENKE, E.
6492
SCHWENKE, G.
6492
SCHWITER, E.J.
5572*
SCOBIE, B.A.
5858
SCOTT, H.W., JR.
5737*, 5851, 5900
SCOTTO, J.
6028*
SCOVILL, W.A.
6125*
SCRUTTON, M.C.
5385*
SEBES, J.I.
5613
SEDASHEVA, IA. E.
5814
SEDMAK, G.V.
6110*
SEDOV, V.M.
6500
SEGURA, J.M.
6062
SEIDLER, D.
5398*
SEIFERT, E.
5671
SEKHON, G.S.
5988
SELANDER, H.G.
5530
SELMAIR, H.
6245*
SELWYN, J.I.
5717
SENAUD, J.
5235
SENECAL, P.
6255
SENGE, T.
6452
SEPPALA, A.
6198
SERA, Y.
5392*, 5405*
SEROR, J.
6552
SERRONI, A.
5385*
SETOGUCHI, T.
6128*
SEUFER, G.
5218
SEWING, K. FR.
5301*
SEYSS, R.
5668
SEZAKI, H.
5226, 5262
SEZILLE, G.
6320
SGRO, J.C.
5400*
SHALIMOV, S.A.
5646
SHARBROUGH, F.W.
6233
SHARMA, B.D.
6575
SHARMA, C.L.N.
5992
SHARMA, O.P.
5796
SHEA, L.
6093*
SHEARMAN, D.J.C.
5901*, 6012*
SHEDRUNOV, V.V.
5290
SHEFER, S.
6128*
SHEPHARD, D.A.E.
6520
SHERLOCK, P.
6516
SHERLOCK, S.
6223*, 6230
SHICHIRI, M.
5629*
SHIDA, T.
5887
SHIELDS, R.
5252*
SHIGETA, Y.
5629*
SHIH, V.E.
6045*
SHIMADA, Y.
5210*
SHIMAJI, T.
5643
SHINDIAIKIN, A.P.
6044
SHINOHARA, M.
6040
SHIOHARA, E.
5406*
SHIRAKI, T.
6559
SHMAKOV, IU. G.
6266
SHMELEV, A.A.
5437
SHMORHUN, S.S.
6379*
SHNIRELMAN, A.I.
5740
SHOJANIA, N.G.
6100*
SHOJI, K.
5622*
SHOOKHOFF, H.B.
6574
SHORT, J.
5555
SHORTER, R.G.
6520
SHPONTAK, A.S.
5646
SHRAER, T.I.
5711
SHUBIN, B.M.
6445

SHUG, A.
5246*
SHULL, H.J.
5737*, 5900
SHUMAN, B.A.L.
5467
SHWACHMAN, H.
5924
SIBOROV, V.S.
5952
SIDDONS, R.C.
5455
SIDORCHENKOV, V.O.
5942
SIEDEK, M.
6396*
SIEGESMUND, K.A.
6110*
SIGRIST-NELSON, K.
5240*
SILK, D.B.A.
5244*
SILLERY, J.
5243*
SILVIS, S.E.
5526*
SIMMONS, R.L.
5537
SIMON, N.M.
5929*
SIMPSON, B.R.
6232
SINGH, H.
5732
SINGH, W.
5988
SINGLETON, J.W.
6257*
SINIAVSKAIA, O.A.
6412
SINKOVICS, J.G.
6484*
SIPPEL, J.
6540
SIRCAR, B.K.
6539
SIZEMOV, O.D.
6417
SJOBERG, N.O.
5206*
SJOVALL, J.
5388*, 5390*
SKACHILOVA, N.N.
6139
SKACHKOV, A.P.
6149
SKALU, I.
5652
SKINNER, D.B.
5605
SKIPPER, A.
5687
SKLIAROV, Y.P.
5325
SKLYANSKAYA, O.A.
5767
SKVORTSOV, M.G.
5873
SLAT, B.
5445
SLEPNEVA, L.B.
5382*
SLOTTER, H.
6392*
SLOVIS, T.L.
6081*
SMEDILE, G.
6182
SMITH, C.Y.
5364*
SMITH, E.
5597*
SMITH, F.
6325
SMITH, F.H.
5261
SMITH, F.W.
5618*
SMITH, I.I.
5521
SMITH, J.E.
5355*
SMITH, J.G., JR.
6358
SMITH, L.B.
5984
SMITH, L.F.
6006
SMITH, P.M.
6224*
SMOLSKY, B.G.
5764
SMOTROVA, I.A.
5763
SMULEWICZ, J.J.
5993
SMYTH, D.H.
5573*
SNODGRASS, W.R.
6218*
SNYDER, M.J.
6525
SOBBE, A.
6392*
SOBOLEV, A.S.
5578
SODUMANN, C.P.
6244*
SOEHENDRA, N.
5889
SOH, C.T.
6563, 6564, 6565, 6566, 6568
SOKOLEWICZ, E.
6250
SOKOLOVA, M.I.
5874
SOLBACH, H.G.
6127*
SOLCIA, E.
5198*
SOLIMAN, L.
6003
SOLOVEI, M.G.
5831
SOLVELL, L.
6479*
SOMMER, U.
5444
SONSINO, E.
5976
SORENSEN, T.I.A.
6107*
SORINOV, A.N.
5970
SORRELL, V.F.
6056*
SOTTO, A.
6304
SOUCHARD, M.
5347
SOUCKOVA, E.
5809
SOULIER, J.P.
6288
SOWINSKA, J.
5653, 5811
SPANGBERG, A.
5996
SPECH, H.J.
6130, 6336*
SPENCER, F.C.
5627*
SPERANZA, V.
5734*
SPICER, F.
6253
SPIEKERMAN, A.M.
6026*
SPINE, G.P.
6334*
SPIVAK, A.M.
5821
SPIVAK, O.M.
5818
SPIVAK, V.P.
5844
SPOHN, K.
6185
SQILLACI, S.
6183
SQUAME, G.
5922
SQUILLACI, S.
6182
SREENIVASAN, A.
5598*
SRIVASTAVA, S.K.
5926
ST. GERICKE, D.
6133
STADALNIK, R.C.
5630*
STADELMANN, O.
6392*, 6396*
STADIL, F.
5307*
STADKOVICH, V.S.
5749
STAFFEN, A.
5958
STAHEL, P.
6558
STALEY, T.E.
5266

STAMM, W.E.
6103*
STANLEY, L.
5223
STANLEY, N.F.
5401*
STANLEY, N.N.
6345*
STARK, J.M.
5600*
STARZL, T.E.
6080*, 6323
STEARNS, M.W.
5692
STECHENBERG, B.W.
6197
STEELE, W.J.
5550
STEFANI, D.V.
6513
STEFANOV, A.V.
6064
STEGAILOV, R.A.
5967
STEGE, T.E.
5250*
STEIGBUEGEL, D.
6240*
STEIGMANN, F.
6195
STEIMAN, I.
5496, 5540
STEIN, T.
6403*
STEINBERG, M.
5687
STEMPINSKI, E.
6423
STENERUD, O.
6174
STENOS, J.
5853
STEPAN, J.
6094*
STEPHENS, D.H.
5863*
STEPHENSON, L.W.
5207*
STETSENKO, G.S.
5832
STEVENS, R.H.
5587
STEWART, J.J.
5269*
STIEHL, A.
6378*, 6386*, 6393*
STIESS, G.
6243*
STIGSBY, B.
6337*
STILLMAN, A.E.
5717
STOCK, C.
5338*
STOKCH, W.
6424
STORER, E.H.
5393*
STORY, J.A.
5538
STOTZNER, H.
6311*
STRAMENTINOLI, G.
5548
STRAND, L.
5850
STRATTA, P.
6295*
STRAUSS, H.W.
5699
STRAUSZER, T.
6547*
STRECKER, E.P.
5635*, 5934*
STREMPLE, J.F.
6515
STRICKLAND, R.G.
6316*
STROHMEYER, G.
6164
STRUNIN, L.
6232
STUART, A.E.
5521
STUBBS, M.
5412*
STUMPE, W.
6083*
STURDEVANT, R.A.L.
5306*
STURZAKER, H.G.
6013*
SUDUKA, P.
5582
SUGA, S.
5643
SUGGETT, A.J.
6021
SUGIHARA, T.
5295, 5503
SUGINO, S.
5503
SUGUIRA, Y.
6209
SUKHAREV, V.F.
5648
SULEMAN, M.I.
5859
SUNDLER, F.
5206*, 5567*
SURI, R.K.
5732
SUSON, E.M.
6193
SUZUKI, H.
6536
SUZUKI, N.
5481
SVENSSON, J.O.
5996
SVIRCHEVICH, A.
5808
SVOBODA, D.J.
6095*
SWAMINATHAN, A.P.
6066
SWARM, R.L.
5386*
SWARTZ, H.
5506
SWEENEY, E.C.
6192
SWEENEY, T.
5224, 6086*
SYNOVETS, A.S.
5742
SYROTA, A.
6350
SZANTO, P.B.
6195
SZENDREI, L.
6528
SZKARADKIEWICZ, A.
6254
SZONN, D.
6420
TABACCHI, L.
6181
TABAKOFF, B.
5539
TABRIZIAN, M.
5965
TADAKI, H.
6202
TAGLIACOZZO, S.
6463
TAGUCHI, K.
5790
TAKADA, T.
6040
TAKAGI, K.
5798
TAKAHASHI, K.
5316
TAKAHASHI, M.
6083*
TAKAHASHI, Y.
6115*
TAKAMATSU, M.
5238*
TAKASAN, H.
5380*
TAKATA, T.
5532
TAKEBAYASHI, S.
6072
TAKEDA, S.
5619*
TAKEUCHI, A.
5205*
TAKEUCHI, K.
5798
TAKEUCHI, N.
5379*
TAKEUCHI, S.
5322
TAKINO, T.
5503
TAMARI, M.
5472
TANAKA, H.
5468
TANAKA, K.
5615*

TANAKA, T.
5354*
TANDLER, B.
5525
TANDON, B.N.
6481*
TANDON, H.D.
5473
TANG, T.T.
6110*
TANGA, M.R.
6020
TANIYAMA, K.
5285
TANNENBAUM, H.
6025*
TANSEY, M.F.
5517
TAPPEL, A.L.
5494
TARASOV, B.M.
6266
TARENZI, L.
5515
TATEMATSU, A.
5226
TAUBMAN, J.
6080*
TAUCHNITZ, C.
6424
TAURA, S.
6072
TAVARTKILADZE, D.A.
6281
TAWIL, T.
5425
TAYLOR, I.K.
5584
TAYLOR, K.J.W.
6224*
TAYLOR, R.M.R.
6402*
TAYLOR, S.L.
5494
TAYLOR, T.V.
5855
TE VELDE, J.
5999
TEDESCO, F.J.
5728, 6008
TEICHER, I.
5993
TEICHMANN, W.
5421
TEIXEIRA, A.
5348
TEIXEIRA, P.A.
5835
TEMIN, V.T.
5646
TEMPLE, J.G.
5802*
TEMPLETON, A.C.
6091*
TENNENBAUM, J.I.
6480*
TEPPER, S.A.
5338
TERNBERG, J.L.
6473
TERROINE, T.
5450
TERZ, J.J.
5936*
TETRUD, J.W.
6593
TEWES, G.
6185
THALER, H.
6297
THAMMIG, R.
6132
THAYSEN, E.H.
6388*
THEODOROPOULOS, G.
6357
THETTER, O.
6442
THEVENOUX, A.M.
6288
THIBERT, F.
5751
THIEL, H.
5397*, 6341*, 6343*
THIELE, D.
6315*
THOMAS, B.J.
6229
THOMAS, J.
5566*
THOMAS, M.
6552
THOMAS, P.J.
5409*
THOMASON, B.M.
6518
THOMPSON, R.A.
6582*
THOMPSON, T.F.
5984
THOMPSON, W.G.
5893
THOR, P.
5333
THORBJARNARSON, B.
6466
THORGEIRSSON, U.P.
6218*
THURSTON, D.G.
6376*
TIKHAIA, P.H.
6411
TIKHII, A.K.
6411
TILAK, T.B.G.
5483
TILSON, M.D.
5224, 5242*, 5571*
TIMBRELL, J.A.
6218*
TIMCHENKO, A.D.
5217
TIMOSHINA, I.V.
5647
TINCKLER, L.F.
6472
TISCORNIA, O.
5337*, 5348
TISCORNIA, O.M.
5287*, 5304*
TITOVA, V.G.
5436
TIZIAN, C.H.
6109*
TOBE, T.
5232
TODA, Y.
6027*
TOFAN, A.V.
5646
TOKARSKAIA, Z.V.
6421
TOLEDO-PEREYRA, L.H.
5537
TOLNAI, K.
6443
TOMASINO, R.M.
6121*
TOMKINS, A.M.
5799*
TONAKI, H.
5614
TONG, M.J.
6249*
TOOYAMA, M.
5502
TORMA, M.J.
5207*, 6075, 6469
TORRANCE, H.B.
5855
TOSKES, P.P.
5205*
TOTH, I.G.
6318*
TOUBOUL, J.P.
5908*
TOULOUKIAN, R.J.
5571*
TOYAMA, T.
5280
TRACK, N.S.
5804, 5839
TRASH, D.B.
5632*
TRAUTWEIN, G.
5398*
TREFFOT, M.J.
5304*
TREHEUX, A.
5677
TREMOLIERES, J.
6321
TRIFONOVA, A.
6532
TRIPATHI, S.N.
5926
TRIPI, S.
6306
TRISHKOVA, L.O.
6234*
TRITAPEPE, R.
6135
TROESCH, V.
5214*

TRULTSCH, D.
6120*
TRUNIN, M.A.
5846
TSAKRAKLIDES, V.
6091*
TSEGA, E.
5895
TSIMMERMAN, Y.S.
5741
TSODIKOV, G.V.
5763
TSUCHIYA, M.
6209
TSUCHIYA, R.
6072
TSUDA, M.
5354*
TSUKADA, K.
5555
TSUKADA, M.
5406*
TSUNODA, T.
6072
TSURUGI, K.
5469
TSUYAMA, S.
5295
TSUZUKI, T.
6209
TSVETKOVA, L.N.
6488
TSYKIN, D.B.
6137
TUBBESING, T.J.
5260
TUCKE, M.
6387*
TUCZEK, H.V.
5407*
TULLIEZ, J.
5543
TURBESSI, G.
6251, 6286
TURCHINI, J.P.
5449
TUREK-MAISCHNEIDER, M.
6085*
TURNBULL, A.C.
6212*
TURNBULL, A.D.
6516
TURTINEN, L.
5787
TUSQUES, J.
5216*, 5334
TUTTON, P.J.M.
5201*
TUYNS, A.J.
5727
TUZHILIN, S.A.
6059, 6070
TVERITINOV, V.N.
5451
TYMPNER, F.
6048*
TYTGAT, G.N.
5921
TZAMALUKAS, G.
5841
UCELLI, S.
6305
UCHIDA, H.
5883
UCHIDA, K.
5379*, 6046*
UCHIYAMA, M.
5253
UDALOV, I.A.
5819
UEZU, J.M.
5622*
UGOLEV, A.M.
5865*
UHL, H.
5218
UHLIG, K.
6221*
ULLMANN, W.
6187
ULLRICH, V.
5558
UMBRUMANTS, O.A.
5807
URAKAMI, Y.
5645
URBANEK, U.
5444
URIBE, P.
6465, 6547*
URSING, B.
6578*
USOVA, N.A.
5765
USPENSKY, V.M.
5748
USVIATSEV, IA. A.
6263
UZBEKOVA, KH. Z.
6268
VAAGE, S.
5719
VAAMONDE, C.A.
6313*
VACHON, A.
6269
VADASZ, T.A.
6144
VAGNE, M.
5321
VAILLE, C.
5347
VAIMAN, M.
5577
VAISMAN, V.D.
5682
VAKIL, U.K.
5598*
VALIRAKHMANOVA, T.D.
5741
VALLÉE, G.
6288
VAN DE VORST, A.
5527
VAN DEN AKKER, M.
6555
VAN EPPS, D.E.
6316*
VAN THIEL, D.H.
5783
VAN WALLEGHEM, J.
5722
VANPARUS, D.F.
6545*
VARCO, R.L.
5894
VARDOULIS, J.
5853
VARLOTEAUX, C.H.
5871
VARMA, R.R.
6110*
VASILIEV, V.S.
6283
VAZQUEZ, V.
6525
VELEV, G.
5826
VENHO, V.M.K.
6401*
VENKATESAN, N.
5550
VENNES, J.A.
5626*
VERAGUTH, P.
6499
VERCELLONE, A.
6295*
VERCESI, G.
5515
VERETENNIKOV, A.L.
6064
VERINE, H.
5337*
VERNIA, P.
5270*
VERTELETSKI, V.V.
5827
VERTENYI, A.
6528, 6530
VERVAT, D.
5722
VESSAL, K.
5934*
VETLUGIN, A.A.
6266
VETTER, W.
5824
VIALLET, A.
6348*
VIDAL, B.C.
5211*
VIDAL, M.T.
6556
VIDAL PLA, R.
6556
VIDO, I.
6228
VIEIRA, J.
6256
VIELLE, G.
6430, 6432
VIJACKI, L.
5943

VIKHROVA, F.K.
5743
VILARDELL, F.
5651
VILASECA, J.
6556
VILLA, F.
6195
VILLIERS, P.
5577
VILOTTE, J.
5976
VINAYAK, V.K.
5557
VINCENT, D.
5338*
VINOGRADOVA, I.L.
6139
VINOT, J.M.
6350
VINTERGALTER, S.F.
W5661
VIRANUVATTI, V.
6236*
VITALI, B.
5515
VITEBSKI, Y.D.
5822
VITI, G.
6471
VIVIEN, P.
6553
VODOLAGIN, V.D.
5763
VOGEL, H.H., JR.
5613
VOGELFANGER, I.J.
5498
VOIMENT, Y.M.
6320
VUKOVICH, V.
5808
VOLKER, K.
6276
VOLOBUEV, N.N.
5772
VON DER DECKEN, A.
5478
VON MAHN, I.
5446
VON MAYERBACH, H.
5542
VON SCHONING, G.
6399*
VOSMIK, J.
5652
VOVOLAGIN, V.D.
5765
VULLIET, F.
5969
VYAS, G.N.
5466
WADDELL, W.G.
5498
WAGENMAKERS, G.
5752
WAGNER, H.N., JR.
5699
WAGNER, R.
5209*, 6394*
WAHLBY, L.
5997
WAHLERS, B.
5715
WAHLIN, T.
5236
WAITZ, J.A.
6576
WAITZ, R.
6184
WALD, A.
5897
WALDMANN, T.A.
5903*, 6117*
WALIA, B.N.S.
6517
WALKER, G.
6055*
WALKER, K.
6376*
WALLACE, A.M.
6249*
WALLACE, R.J.
5293
WALLENSTEIN, F.
6185
WALLER, S.L.
6482*
WALSH, D.A.
5495
WALSH, J.H.
5685, 6305, 6483*
WALTON, B.
6232
WANSON, J.C.
5373*, 5375*
WARD, K.M.
6223*
WARNATZ, H.
6298, 6506
WARREN, K.W.
6519
WARRICK, M.W.
5308*
WASHINGTON, S.L.A.
5364*
WASSERMAN, A.E.
5523
WATANABE, H.
5249*, 5595*, 5615*, 5622*
WATANABE, K.
5324
WATARI, N.
5504
WATSON, W.W.
5794
WATTS, G.T.
6474
WEBB, D.I.
6103*
WEBB, W.
5900
WEBER, M.
6370
WEBER, P.
5558
WEDEL, F.P.
5378*
WEERASINGHE, W.M.T.
5258
WEGMANN, R.
5329
WEGMANN, T.
6557
WEHLING, H.
5889
WEHMEYER, D.L.
6541
WEHRMANN, M.
6275
WEIDENHILLER, S.
5631*
WEIDNER, F.
5905*
WEILL-BOUSSON, M.
6552
WEILL, F.
5636*
WEINBREN, K.
5364*
WEINER, H.
5318
WEIS, H.J.
5358*, 6413
WEISMANN, R.E.
5899
WEISS, M.
6340*
WEISS, Y.
6577*
WEITZEL, D.
6398*
WELTE, D.
6342*
WENZEL, M.
5884
WERNER, B.
6248*
WERNZE, H.
6130
WESEL, A.J.T.
6545*
WESER, E.
5425
WESSELER, E.P.
5223
WESSELER, T.
5756
WESTON, M.J.
6089*
WEX, P.
6406
WEXLER, M.J.
6347*
WHEELER, G.E.
5452
WHELAN, G.
5411*
WHELDRAKE, J.F.
5505
WHELTON, M.
6360
WHITE, R.R., IV
5782

WHITE, I.T.
6054*
WHITEHEAD, W.A.
5857
WHITFIELD, A.E.
5241*
WHITTLE, H.C.
5904*
WHYTE, A.S.
5854
WICKS, R.C.
6237*
WICKSMAN, K.S.
5621*
WIEGELMANN, W.
6127*
WIERZBOLOWICZ, J.
5653, 5811
WIESE, W.
6245*
WIGHT, D.G.D.
6522
WILDERINK, F.
5921
WILDGRUBE, H.J.
6105*
WILK, P.
5586
WILKINS, E.W., JR.
5720
WILKINSON, P.
6331
WILLERS, H.
6275
WILLGEROTH, C.
6492
WILLIAMS, A.O.
5990
WILLIAMS, D.M.J.
6224*
WILLIAMS, H.T.G.
5892
WILLIAMS, L.F., JR.
5987
WILLIAMS, M.J.
6580*
WILLIAMS, R.
6089*, 6238*
WILLIAMS, R.C., JR.
6316*
WILLMAN, V.L.
5381*
WILLSON, R.A.
6239*
WILSON, F.A.
5247*
WILSON, S.D.
5286*
WILSON, S.E.
5599*
WINAND, J.
5350
WINGATE, D.L.
5259
WINSTON, M.
5599*
WISBEY, M.L.
5465
WISE, L.
6403*
WITTENBERG, J.
5987
WOEBER, K.
6324
WOHLGEMUTH, B.
5544, 6104*
WOJCZUK, D.
5925
WOJTOWICZ, M.
6439
WOLF, B.S.
5617*
WOLFERT, W.
5448
WOLFF, R.
5413*
WOLFMAN, E.F., JR.
5860*, 5861*
WOLLENBERG, P.
5558
WOLPERS, C.
6440
WOOD, C.B.
6034
WOOD, J.D.
5274*
WOODWARD, E.R.
5729
WOODWARD, T.E.
6525
WOODWARD, W.E.
6486*
WOOLF, D.
6222*
WORTH, M.H.
5983
WOZIWODZKI, G.
5954
WRIGHT, C.B.
5585
WRIGHT, H.K.
5224, 5580
YAJIMA, G.
5485, 5501, 5502
YAMADA, A.
6040
YAMADA, E.
5786
YAMADA, T.
5380*, 5389*
YAMAGATA, S.
5622*, 6202
YAMAGUCHI, K.
5948
YAMAGUCHI, M.
5468, 6188
YAMAGUCHI, T.
5232
YAMAMATO, M.
5658
YAMAMOTO, M.
5888
YAMAMOTO, T.
6524
YAMAMURA, Y.
5379*, 6115*
YAMAOKA, Y.
5389*
YAMAZAKI, M.
5888
YANBE, A.
6202
YARDLEY, J.J.
5723
YASHIMA, K.
5238*
YEUNG, S.K.F.
5493, 5540
YLIKORKALA, O.
6108*
YLOSTALO, P.
6108*
YOKOYAMA, H.
5788
YOKOYAMA, I.
5280
YOSHIDA, M.
5629*
YOSHIDA, T.
5615*
YOSHINO, R.
6072
YOSHIZAWA, S.
5229
YOSIZAWA, Z.
5481
YOUN, K.H.
5502
YOUNG, M.
5584
YOUNG, S.R.
5417*
YOUNGER, R.K.
5900
YOUSEF, I.M.
5457
YRJOLA, H.
6226
YU, F.L.
5556
ZABELINSKII, E.K.
5865*
ZACCHEROTTI, L.
5657
ZACHERL, H.
5958
ZAJEC, M.
5278
ZAKI, A.E.
5977
ZAKREVSKII, N.N.
6284
ZALEWSKI, H.
5836
ZALEWSKY, C.
5207*
ZAMCHECK, N.
6033
ZANGL, A.
6425
ZARINS, C.K.
5605
ZARIVCHATSKII, M.F.
6501

ZARZOVA, E.
6079*
ZASLAVSKAIA, E.S.
6411
ZATOLOKIN, V.S.
6151
ZAVERI, M.P.
5848
ZELDER, O.
5447
ZEMSKOV, N.N.
6415
ZETTERMAN, R.
6325, 6328
ZETTERMAN, R.K.
6312*
ZHAMSARANOVA, S.D.
5578
ZHITNJUK, R.I.
6437
ZHIVANOVICH, M.
5808
ZHUKOVSKAIA, E.S.
6037
ZIMMERMANN, A.
6498
ZIMMERMANN, P.
6422
ZIMOVSKY, V.L.
6436
ZINCHENKO, T.V.
6379*
ZINEVICH, V.P.
5806
ZINNER, M.J.
5787
ZIVA, V.K.
6262
ZIZKOVSKY, V.
6094*
ZMUDZINSKI, J.
5925
ZU DOHNA, P.
6294*
ZUCKERMAN, A.J.
6241*
ZUIDEMA, G.D.
6049*
ZWIERZ, J.
6431

NUMBERS FOLLOWED BY AN ASTERISK INDICATE ABSTRACTS. OTHER NUMBERS REFER TO CITATIONS.

ABDOMEN
CALCIFICATION
DISEASES ASSOCIATED WITH, 5778
IRRADIATION
SMALL INTESTINE, 5581
MUSCULOSKELETAL SYSTEM
ANOMALY, CONGENITAL, 6496
PAIN
SEDATIVES, 5882
TRAUMA
PERITONEOSCOPY, 5625*
PERITONITIS, 5625*

ABSCESS
LIVER
AMEBIASIS, 6549*, 6551*, 6571
DIAGNOSIS, 6170
DRAINAGE, 5627*
DRUG TREATMENT, 6551*
HEPATECTOMY, 6152
PERFORATION, 6132
RADIOLOGY, 6132
RECURRENCE, 6571
SCANNING, SCINTILLATION, 5627*
SURGERY, 6170
PANCREAS
PSEUDOCYST, 6044

ABSORPTION
ALCOHOL
DRUG EFFECTS ON, 5254
GASTROINTESTINAL TRACT, 5254
AMINO ACIDS
POLYPHENOLS, 5261
SMALL INTESTINE, 5253, 5261
VITAMIN D, 5253
CALCIUM
DEFICIENCY, 5263
DIETARY FACTORS, 5263
SMALL INTESTINE, 5263
CHOLINE
SMALL INTESTINE, 5238*
GLUCOSE
GASTRECTOMY, 5774
VAGOTOMY, 5774
GLYCOSIDE
MALNUTRITION, 6485*
MICROORGANISMS, 6485*
ILEUM
ILEOSTOMY, 5867*
INFLAMMATORY BOWEL DISEASE, 6520
PERFUSION, 5249*
TECHNIQUES, 5249*
INTESTINE
CHYMOTRYPSIN, 5342*
JEJUNUM
FATTY ACIDS, 5246*
OBSTRUCTION, 5580
PEPTIDES, 5239*
WATER, 5246*
LIPIDS
GASTRECTOMY, 5774
VAGOTOMY, 5774
NUCLEOTIDES
SMALL INTESTINE, 5238*
PANCREAS
ENZYMES, 5342*
PEPTIDES
SMALL INTESTINE, 5244*

ABSORPTION (continued)
POTASSIUM
CELIAC DISEASE, 5910*
PROTEIN
INTESTINE, 5266
NEONATE, 5266
SMALL INTESTINE
SUGAR, 5251*
STOMACH
ASPIRIN, 5258
BILE ACIDS, 5255
CARBENOXALONE, 5258
VITAMIN B12
BLIND LOOP SYNDROME, 5902*
GASTRECTOMY, 5759
ILEOSTOMY, 5866*
JEJUNUM, 5902*
SMALL INTESTINE, 5598*
VITAMIN D
DEFICIENCY, 5253
SMALL INTESTINE, 5387*
WATER
CIRRHOSIS, 6340*
PHYSICAL FACTORS, 6340*
XYLOSE
CIRCULATION, 5925

ACETYLCHOLINE
SMALL INTESTINE
MOTILITY, 5267*
SYNTHESIS
NERVOUS CONTROL, 5291
SALIVARY GLANDS, 5291

ACHALASIA
DILATATION, 5707
SURGERY, 5721

ACHLORHYDRIA
SEE ALSO ACID SECRETION

ACID PHOSPHATASE
SEE PHOSPHATASE, ACID

ACID SECRETION
SEE ALSO ACHLORHYDRIA, HYPERCHLORHYDRIA, HYPOCHLORHYDRIA
SEE ALSO HYPERCHLORHYDRIA
SEE ALSO HYPOCHLORHYDRIA
STOMACH
ACIDITY, 5310*
ALCOHOL, 5287*, 5302*
ANTISECRETORY AGENTS, 5315
ANTRECTOMY, 5312*
CALCITONIN, 5769
CALCIUM, 5307*, 5310*
CHOLERA, 5601
DRUG EFFECTS ON, 5312*, 5842
FEEDING, 5313
GASTRIN, 5321, 5322
H2 RECEPTOR ANTAGONISTS, 5309*, 5324
HEPATECTOMY, 5297*
HISTAMINE, 5298*, 5309*, 5324, 5328, 5734*
HORMONE CONTROL, 5296*
INTESTINE, 5325
KIDNEY TRANSPLANTATION, 5751
MAGNESIUM, 5307*
NERVOUS CONTROL, 5287*

ACID SECRETION (continued)
STOMACH (continued)
PANCREAS, 5325
PANCREATITIS, CHRONIC, 6059
PARASYMPATHOMIMETIC AGENTS, 5298*
PEPTIC ULCER, 5807
PROSTAGLANDIN, 5381*
PROTEIN, 5313
PYLOROPLASTY, 5312*
SECRETIN, 5322
STEROID, 5649
VAGOTOMY, 5298*, 5312*, 5734*, 5849

ACIDITY
BLOOD
GASTRIN, 5306*, 5310*
DUODENUM
CHOLECYSTOKININ, 5574*
MOTILIN, 5273*
GASTROINTESTINAL
ILEUS, 5877
STOMACH
ACID SECRETION, 5310*
GASTRECTOMY, 5825
PEPTIC ULCER, 5825
PERMEABILITY, 5265
URINE
LIVER DISEASE, ALCOHOLIC, 6313*

ACIDOSIS
CIRRHOSIS, 6354

ADENOSINE TRIPHOSPHATASE
BILE DUCT
ULTRASTRUCTURE, 5368*
JEJUNUM
BILE ACIDS, 5241*
LIVER
HYPOCHOLESTEREMIC AGENTS, 5531

ADENYL CYCLASE
COLON
TRANSPORT, 5204*
ULTRASTRUCTURE, 5204*
STOMACH
PENTAGASTRIN, 5301*
STARVATION, 5301*

ADHESIVES
SEE TISSUE ADHESIVES

ADOLESCENTS
SEE ALSO AGE FACTORS
CROHNS DISEASE
DIAGNOSIS, 6579*
PEPTIC ULCER
EPIDEMIOLOGY, 5811

ADRENERGIC AGENTS
SMALL INTESTINE
MOTILITY, 5267*
STOMACH
CIRCULATION, 5597*

ADRENERGIC BLOCKING AGENTS
LIVER
GLUCONEOGENESIS, 5484
STOMACH
EROSIONS, 5798
SECRETION, 5798

AFFERENT LOOP SYNDROME
PANCREATITIS
DISEASES ASSOCIATED WITH, 6067

AFLATOXIN
METABOLISM
LIVER, 5524
PRIMATES, 5524
NEOPLASMS, MALIGNANT
BILIARY, 5483

AGE FACTORS
SEE ALSO CHILDREN, NEONATE, ADOLESCENTS
ANTRUM
GASTRIN, 5206*
APPENDIX
MORPHOLOGY, 5203*
CHOLELITHIASIS
PROGNOSIS, 6422
THERAPY, 6468
DUODENUM
GASTRIN, 5206*
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 6250
LIVER FUNCTION TESTS, 6281
IMMUNOGLOBULINS
SMALL INTESTINE, 5604
LACTOSE TOLERANCE, 5924
LIVER
REGENERATION, 5534
LIVER INJURY
CYTOCHROMES, 5485
PANCREAS
GASTRIN, 5206*
PANCREATITIS
COMPLICATIONS, 6062
ETIOLOGY, 6062
STOMACH
ENDOCRINE SYSTEM, 5334
SECRETION, 5330
ULTRASTRUCTURE, 5334

ALBUMIN
BLOOD
DIETARY FACTORS, 5456
STARCHES, 5456
SUCROSE, 5456
INTESTINE
PARASITES AND PARASITIC DISEASE, 6564
LIPIDS
SMALL INTESTINE, 5600*
LIVER DISEASE
AUTOIMMUNITY, 6082*
LIVER INJURY
GALACTOSAMINE, 5446
METABOLISM
GASTROINTESTINAL, 5293
LIVER, 5402*
STOMACH
NEOPLASMS, MALIGNANT, 5770

ALCOHOL
ABSORPTION
DRUG EFFECTS ON, 5254
GASTROINTESTINAL TRACT, 5254
AMINO ACIDS
SYNTHESIS, 5412*

ALCOHOL (continued)
BILE ACIDS
METABOLISM, 5388*
CIRRHOSIS
ETIOLOGY, 6318*
RISK FACTORS, 6339*
CYTOCHROMES
LIVER, 5406*
DEHYDROGENASE
LIVER, 5528
FATTY LIVER
TRIGLYCERIDE, 5376*
GASTRIN
SECRETION, 5348
GASTROINTESTINAL
MOTILITY, 5254
HYPERLIPOPROTEINEMIA
SIMULATION, 5414*
LIPIDS
METABOLISM, 5414*
LIVER
DRUG METABOLISM, 5558
ENZYMES, 5377*
PATHOLOGY, 5377*
ULTRASTRUCTURE, 5513
LIVER INJURY
DRUG TREATMENT, 5548
METABOLISM
ALCOHOLISM, 6315*
COENZYMES, 6315*
FEEDING, 6315*
LIVER, 5487, 5489
LIVER INJURY, 5423
PHENOBARBITAL, 6315*
OXIDATION
LIPIDS, 5557
LIVER, 5557
MITOCHONDRIA, 5557
PANCREAS
SECRETION, 5287*, 5348
PANCREATITIS
ETIOLOGY, 6049*
TRIGLYCERIDE, 6049*
PROTEIN
SYNTHESIS, 5372*
STOMACH
ACID SECRETION, 5287*, 5302*
CIRCULATION, 5590*
CYCLIC ADENOSINE MONOPHOSPHATE, 5302*
TOLERANCE
SIMULATION, 5367*
TRIGLYCERIDE
METABOLISM, 5376*

ALCOHOLIC LIVER DISEASE
SEE LIVER DISEASE, ALCOHOLIC

ALCOHOLISM
ALCOHOL
METABOLISM, 6315*
BLOOD
GASTRIN, 5304*
CIRRHOSIS
IMMUNOLOGY, 6328
REVIEWS, 6326
ETIOLOGY
ENVIRONMENTAL FACTORS, 5361*
GENETIC FACTORS, 5361*

ALCOHOLISM (continued)
FATTY LIVER
REVIEWS, 6327
HEMOCHROMATOSIS
REVIEWS, 6329
HEPATITIS, NONVIRAL
IMMUNOLOGY, 6328
IMMUNOLOGY
REVIEWS, 6332
LIVER
NEOPLASMS, MALIGNANT, 6195
LIVER DISEASE
REVIEWS, 6331
PANCREAS
HYALIN, 6052*

ALCOHOLS, POLYHYDRIC
LIVER
LIPOPROTEINS, 5430

ALDEHYDES
DEHYDROGENASE
LIVER, 5539

ALDOLASE
HEPATITIS, VIRAL
DIAGNOSIS, 6268

ALKALINE PHOSPHATASE
SEE PHOSPHATASE, ALKALINE

ALKALOIDS
PANCREAS
CYTOTOXICITY, 5208*
PROTEIN
SECRETION, 5374*
STOMACH
MOTILITY, 5284

ALKALOSIS
CIRRHOSIS, 6354

ALKLATING AGENTS
INTESTINE
DRUG EFFECTS ON, 5584

ALLERGY
DIETARY FACTORS
GASTROENTERITIS, 6480*
SMALL INTESTINE
RADIOLOGY, 5888
ULCERATIVE COLITIS
DISEASES ASSOCIATED WITH, 6011*

AMEBIASIS
ABSCESS
LIVER, 6571
COLON
DISEASES ASSOCIATED WITH, 6552
NECROSIS, 5992
PERFORATION, 5992
PSEUDOTUMOR, 6007
IMMUNOLOGY
TECHNIQUES, 6566
LARGE INTESTINE
DIAGNOSIS, 6558
NECROSIS, 6575
THERAPY, 6505

AMEBIASIS (continued)
LIVER
ABSCESS, 6549*, 6551*
DIAGNOSIS, 6549*, 6558
DISEASES ASSOCIATED WITH, 6552
THERAPY, 6158
SMALL INTESTINE
NECROSIS, 6575
THERAPY, 6546*

AMINO ACIDS
ABSORPTION
POLYPHENOLS, 5261
SMALL INTESTINE, 5253, 5261
VITAMIN D, 5253
BILE ACIDS
BINDING, 5457
CIRRHOSIS
MALNUTRITION, 6366
FATTY LIVER
DRUG-INDUCED, 5474
LIVER
CARBON TETRACHLORIDE, 5419*
METABOLISM
DIETARY FACTORS, 5468
HEPATITIS, VIRAL, 6283
LIVER, 5468, 5476, 5522
PANCREATITIS
BLEEDING, 5340*
FAMILIAL FACTORS, 6045*
SYNTHESIS
ALCOHOL, 5412*
LIVER, 5412*
URINE
PANCREATITIS, 6045*

AMMONIA
METABOLISM
LIVER COMA, 5462

AMPULLA OF VATER
NEOPLASMS, MALIGNANT, 6466
PANCREATECTOMY, 6519

AMYLASE
PANCREAS
CALCULI, 6027*
GENETICS, 5344*
PEPTIC ULCER
PERFORATION, 5854
SECRETION
CALCIUM, 5350
PANCREAS, 5350

ANALGESICS
LIVER
DRUG METABOLISM, 5451
LIVER INJURY
REVIEWS, 6231
STOMACH
PATHOLOGY, 5308*

ANEMIA
DISEASES ASSOCIATED WITH
CIRRHOSIS, 6371
PARASITES AND PARASITIC DISEASE
DISEASES ASSOCIATED WITH, 6562

ANEMIA, PERNICIOUS
ETIOLOGY, 5760
PANCREAS
SECRETION, 6029*

ANESTHETICS
HEPATITIS, NONVIRAL
DRUG-INDUCED, 6226, 6230
LIVER INJURY
DRUG-INDUCED, 5518, 6232

ANEURYSM
DUODENUM
ARTERY, 6542
STOMACH
ARTERY, 6542

ANGIOGRAPHY
BILIARY
BLEEDING, 6434
COMPLICATIONS, 6434
TECHNIQUES, 6210*
BILIARY DISEASE
TECHNIQUES, 5643, 5645, 5646
CIRRHOSIS, 6363
BLEEDING, 6362
SURGERY, 6362
GALLBLADDER
PERFORATION, 6452
GALLBLADDER DISEASE
TECHNIQUES, 6472
JAUNDICE, OBSTRUCTIVE
TECHNIQUES, 5701
LIVER
ECHINOCOCCOSIS, 5677
NEOPLASMS, MALIGNANT, 5696, 6083*
TECHNIQUES, 5644
PANCREAS
CYSTS, 6040
NEOPLASMS, MALIGNANT, 5639*
PANCREAS DISEASE, 6039
REVIEWS, 6383*
TECHNIQUES, 5645, 5646
PANCREATITIS, CHRONIC, 5639*
PAPILLA OF VATER, 5676
PORTAL HYPERTENSION, 6347*, 6360
SMALL INTESTINE
NEOPLASMS, BENIGN, 5655
NEOPLASMS, MALIGNANT, 5655

ANILINE
LIVER
DRUG METABOLISM, 5451

ANOMALY
DUODENUM
ARTERY, 5606
ESOPHAGUS
ENDOSCOPY, 5728
PANCREAS
ARTERY, 5606
PYLORUS
PEPTIC ULCER, 5755

ANOMALY, CONGENITAL
ABDOMEN
MUSCULOSKELETAL SYSTEM, 6496
ANORECTUM
DEFECATION, 5638*

ANOMALY, CONGENITAL (continued)
BILE DUCT
DILATATION, 6415
LIVER, 6109*
COLON, 5962
GASTRECTOMY, 5954
GALLBLADDER, 6431
LIVER
BILE DUCT, 6396*
CYSTS, 6121*
FIBROSIS, 6121*
MEGACOLON
CHILDREN, 5953
NEONATE, 5953

ANORECTUM
ANOMALY, CONGENITAL
DEFECATION, 5638*
CIRCULATION
MORPHOLOGY, 5608
SURGERY, 5608

ANOXIA
NUCLEIC ACIDS
LIVER, 5427

ANTACIDS
GASTRIN
BLOOD, 5838
GASTROINTESTINAL
BLEEDING, 6479*

ANTIARRHYTHMIC AGENTS
LIVER INJURY
DRUG-INDUCED, 6219*

ANTIBACTERIALS
HEPATITIS, NONVIRAL
DRUG-INDUCED, 6217*
LIVER
DRUG METABOLISM, 6218*
LIVER INJURY
DRUG-INDUCED, 6218*
SALMONELLOSIS
DRUG TREATMENT, 6540
ULCERATIVE COLITIS
DRUG TREATMENT, 6019, 6023

ANTIBIOTICS
SEE ALSO ANTIBACTERIALS, DRUG TREATMENT
BILIARY
SURGERY, 6474
BILIARY TRACT
INFECTION, BACTERIA, 6424
CHOLANGITIS
TECHNIQUES, 6443
CHOLECYSTITIS
TECHNIQUES, 6443
CHOLERA
DRUG TREATMENT, 6539
COLITIS
DRUG-INDUCED, 5930*, 6008
ENTEROCOLITIS
DRUG-INDUCED, 6534
EXCRETION
BILE, 6401*
FATTY LIVER
DRUG-INDUCED, 5559

ANTIBIOTICS (continued)
GASTRITIS
DRUG TREATMENT, 5743
HELMINTHIASIS
DRUG EFFECTS ON, 6576
LIVER
PATHOLOGY, 6223*
PHOSPHORYLATION, OXIDATIVE, 5443
LIVER INJURY
DRUG-INDUCED, 6223*
PANCREAS
SECRETION, 5343*
PROTEIN
SYNTHESIS, 5343*
PROTOZOA
DRUG EFFECTS ON, 6576
SALMONELLOSIS, 6525
CARRIER STATE, 6512
DRUG TREATMENT, 6540

ANTICHOLINERGIC AGENTS
GASTRITIS, ATROPHIC
SECRETION, 5741
IRRITABLE COLON, 5982
PEPTIC ULCER
DRUG TREATMENT, 5821
STOMACH
SECRETION, 5812, 5829

ANTICOAGULANTS
BILE
EXCRETION, 5453
LIVER
DRUG METABOLISM, 5453

ANTICONVULSANTS
FOLIC ACID
METABOLISM, 5482

ANTIDIABETIC AGENTS
LIVER
DRUG METABOLISM, 6150

ANTIDIARRHEALS
CHOLERA
DRUG TREATMENT, 6539
ESOPHAGUS
MOTILITY, 5277

ANTIEMETICS
GALLBLADDER
MOTILITY, 6467
STOMACH
MOTILITY, 5738*
SECRETION, 5738*

ANTIENZYMES
LIVER
ISCHEMIA, 6133
PANCREATITIS
DRUG TREATMENT, 6065, 6073

ANTIGEN
METABOLISM
LIVER, 5542

ANTIGEN, AUSTRALIA
CARRIER STATE
IMMUNITY, 6237*

ANTIGEN, AUSTRALIA (continued)
CIRRHOSIS, 6236*, 6245*
CHILDREN, 6246*
HEPATITIS, CHRONIC, 6245*
COMPLEMENT, 6290*
CORTICOSTEROIDS, 6306
KIDNEY DISEASE, 6295*
HEPATITIS, SERUM
CHILDREN, 6260
HEPATITIS, VIRAL, 6245*
AGE FACTORS, 6250
CARRIER STATE, 5466
CHILDREN, 6260
DIAGNOSIS, 6278
EPIDEMIOLOGY, 6253
IMMUNITY, 6237*
IMMUNOLOGY, 5466, 6279
LIVER FUNCTION TESTS, 6274
REVIEWS, 6277
RISK FACTORS, 6242*
SEROLOGICAL DIAGNOSIS, 6276
IMMUNOLOGY, 6271
CIRRHOSIS, 6273
HEPATITIS, CHRONIC, 6273
HEPATITIS, VIRAL, 6273
LIVER
CARRIER STATE, 6251, 6286
HEPATITIS, VIRAL, 6251, 6286
NEOPLASMS, MALIGNANT, 6138, 6143, 6195, 6236*, 6304, 6357
LIVER DISEASE, 6094*
METABOLISM
CARRIER STATE, 6288
HEPATITIS, CHRONIC, 6286
NUCLEIC ACIDS
SYNTHESIS, 6244*

ANTIGEN, CARCINOEMBRYONIC
COLON
NEOPLASMS, MALIGNANT, 5935*
GASTROINTESTINAL
NEOPLASMS, MALIGNANT, 5688
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 6013*
PANCREAS
NEOPLASMS, MALIGNANT, 6033
ULCERATIVE COLITIS, 6014*
NEOPLASMS, MALIGNANT, 6013*

ANTIHORMONES
LIVER FUNCTION TESTS
DRUG EFFECTS ON, 5541

ANTIINFLAMMATORY AGENTS
GASTROINTESTINAL
BLEEDING, 6492
LIVER INJURY
DRUG-INDUCED, 6222*
STOMACH
PATHOLOGY, 5308*

ANTIMETABOLITES
LIVER INJURY
DRUG-INDUCED, 5294
PANCREAS
PATHOLOGY, 5294
SMALL INTESTINE
PATHOLOGY, 5294

ANTINEOPLASTIC AGENTS
JEJUNUM
KINETICS, CELL, 5589
LIVER
DRUG METABOLISM, 5295
PANCREAS
PATHOLOGY, 5208*
TRYPSIN, 5609
SALIVARY GLANDS
DRUG METABOLISM, 5295
SMALL INTESTINE
DRUG METABOLISM, 5295

ANTISECRETORY AGENTS
STOMACH
ACID SECRETION, 5315
SECRETION, 5329

ANTITRYPSIN, ALPHA
DEFICIENCY
CIRRHOSIS, 6333*
DISEASES ASSOCIATED WITH, 6087*
LIVER, 6098*
LIVER DISEASE, 6145
LIVER
NEOPLASMS, MALIGNANT, 6087*
PATHOLOGY, 6098*

ANTRECTOMY
SEE ALSO GASTRECTOMY
SEE ALSO STOMACH, SURGERY, GASTRECTOMY
STOMACH
ACID SECRETION, 5312*
VAGOTOMY, 5851

ANTRUM
SEE ALSO STOMACH
ENDOSCOPY, 5695
GASTRIN
AGE FACTORS, 5206*
EMBRYOLOGY, 5206*
STOMACH
SECRETION, 5335

ANUS
SEE ALSO LARGE INTESTINE
NEOPLASMS, BENIGN
TRANSFORMATION, 5976
NEOPLASMS, MALIGNANT
SURGERY, 5969

APPENDICITIS
CROHNS DISEASE
DISEASES ASSOCIATED WITH, 6577*
DIAGNOSIS, 5966, 5997
LEUKOCYTE
PHAGOCYTOSIS, 5944
SEASONAL FACTORS, 5957
SURVIVAL
RISK FACTORS, 5979

APPENDIX
SEE ALSO LARGE INTESTINE
CARCINOIDS
SURGERY, 5970
INFILTRATION
SURGERY, 5951
THERAPY, 5951

APPENDIX (continued)
MORPHOLOGY
AGE FACTORS, 5203*
LYMPHATICS, 5203*
NEOPLASMS
PATHOLOGY, 5977
POLYPS
RADIOLOGY, 5958
ULCER
DISEASES ASSOCIATED WITH, 5995

APPETITE
HORMONE CONTROL
CHOLECYSTOKININ, 5603

ARTERY
DUODENUM
ANEURYSM, 6542
ANOMALY, 5606
PANCREAS
ANOMALY, 5606
STOMACH
ANEURYSM, 6542

ASCITES
CIRRHOSIS
CIRCULATION, 6349*
PORTAL HYPERTENSION, 6351
REVIEWS, 6359
THERAPY, 6351
LIVER
REGENERATION, 6080*
SMALL INTESTINE
PNEUMATOSIS, 5895

ASPIRIN
CHOLERESIS
DRUG-INDUCED, 5431
GASTROINTESTINAL
BLEEDING, 6154, 6479*
GLUCOSAMINE
METABOLISM, 5505
LIVER INJURY
LIVER FUNCTION TESTS, 6154
REYES SYNDROME, 6093*
STOMACH
ABSORPTION, 5258
BIOPOTENTIALS, 5258
CIRCULATION, 5590*
EROSIONS, 5798
PATHOLOGY, 5308*
SECRETION, 5798

ATHEROSCLEROSIS
LIPIDS
METABOLISM, 5538
LIVER FUNCTION TESTS
HYPERTENSION, 6155
MALABSORPTION
DISEASES ASSOCIATED WITH, 5922

ATRESIA
BILE DUCT
DOWNS SYNDROME, 6190
PATHOLOGY, 6211*
PROGNOSIS, 6211*
BILIARY
DRUG TREATMENT, 6227
LIPOPROTEINS, 5624*

ATRESIA (continued)
BILIARY (continued)
PATHOLOGY, 6381*
PHENOBARBITAL, 6227
ESOPHAGUS
DISEASES ASSOCIATED WITH, 5724
FAMILIAL FACTORS, 5724
RADIOLOGY, 5722, 5733
SURGERY, 5719, 5722

ATROPHY
JEJUNUM
CELIAC DISEASE, 5921

ATROPINE
INTESTINE
DRUG EFFECTS ON, 5584

AUTOIMMUNITY
CIRRHOSIS, BILIARY
ETIOLOGY, 6298
REVIEWS, 6101*
GASTRITIS, 5761
HEPATITIS
DRUG-INDUCED, 6101*
HEPATITIS, CHRONIC
REVIEWS, 6101*
HEPATITIS, VIRAL
REVIEWS, 6101*
LIVER
RUPTURE, 6198
LIVER DISEASE
ALBUMIN, 6082*
IMMUNOGLOBULINS, 6082*

BACTERIA
BILE
CHOLECYSTITIS, 6465
ILEUM
ILEOSTOMY, 5867*
LARGE INTESTINE
CHOLESTEROL, 5928*
POLYPS, 5928*

BALANCE STUDIES
HEPATITIS, VIRAL
TRACE ELEMENTS, 6234*

BEZOARS
SEE FOREIGN BODIES

BILE
ANTIBIOTICS
EXCRETION, 6401*
BACTERIA
CHOLECYSTITIS, 6465
BILE ACIDS
CHOLECYSTITIS, 6454
CHOLESTASIS, 6387*
CIRCADIAN RHYTHM, 6387*
BILIRUBIN
GLYCOSIDES, 6213*
CHEMICAL COMPOSITION
CHOLECYSTECTOMY, 6376*
GLYCOPROTEINS, 5481
CHOLESTEROL
CHENODEOXYCHOLIC ACID, 5366*
EXCRETION, 5424

BILE (continued)
EXCRETION
ANTICOAGULANTS, 5453
BILE ACIDS, 6102*
CARBON TETRACHLORIDE, 5500
ESTROGENS, 5459
SULFOBROMOPHTHALEIN, 5410*
GLUTAMYL TRANSPEPTIDASE, GAMMA
CHOLESTASIS, 6387*
LIPIDS
CHENODEOXYCHOLIC ACID, 5366*
PHENOBARBITAL, 5442
VITAMIN C, 5371*
PANCREAS
CALCULI, 6027*
PEPTIDES, 5429
PHOSPHATASE, ALKALINE
CHOLESTASIS, 6387*
CIRCADIAN RHYTHM, 6387*
PHOSPHOLIPID, 5429
EXCRETION, 5424
SECRETION
ENTERECTOMY, 5425
PEPTIC ULCER, 5820
PROSTAGLANDIN, 5381*
REVIEWS, 5434
SECRETIN, 5445
SPHINCTER
NERVOUS CONTROL, 5517
PRESSURE STUDIES, 5517
STOMACH
SECRETION, 5305*
SULFOBROMOPHTHALEIN
EXCRETION, 5411*
PHENOBARBITAL, 5411*

BILE ACIDS
BILE
CHOLECYSTITIS, 6454
CHOLESTASIS, 6387*
CIRCADIAN RHYTHM, 6387*
EXCRETION, 6102*
BINDING
AMINO ACIDS, 5457
CHOLECYSTECTOMY, 6405*
DRUG EFFECTS ON, 6405*
BLOOD
HYPERBILIRUBINEMIA, 6118*
LIVER DISEASE, 5632*, 6118*, 6124*
CHOLELITHIASIS
ETIOLOGY, 6413
THERAPY, 6413
CHOLESTEROL
EXCRETION, 5424
COLON
BIOPOTENTIALS, 5572*
MORPHOLOGY, 5245*
MOTILITY, 5245*
SECRETION, 5572*
DETOXIFICATION, 6393*
INTESTINE
DISEASES, 6497
JEJUNUM
ADENOSINE TRIPHOSPHATASE, 5241*
TRANSPORT, 5241*, 5248*
LIVER
ANALYSIS, 6124*
CHEMICAL COMPOSITION, 5552
LIVER DISEASE, 6124*
METASTASES, 6105*

BILE ACIDS (continued)
LIVER DISEASE
DIAGNOSIS, 5632*, 6118*
METABOLISM
ALCOHOL, 5388*
CARBON TETRACHLORIDE, 5480
CHENODEOXYCHOLIC ACID, 6378*, 6386*, 6393*
CHOLELITHIASIS, 5403*, 6386*
CHOLESTASIS, 6120*
CIRRHOSIS, 6105*
CYTOCHROMES, 6120*
DIETARY FACTORS, 5403*
DRUG EFFECTS ON, 6393*
JAUNDICE, OBSTRUCTIVE, 6105*
KINETICS, 5409*
LIVER, 5388*, 5403*, 5480, 6102*, 6120*
PHENOBARBITAL, 5480
PHOSPHOLIPID
EXCRETION, 5424
SECRETION
LIVER, 5353*
STOMACH
ABSORPTION, 5255
CIRCULATION, 5590*
EROSIONS, 5255
SYNTHESIS
CIRCADIAN RHYTHM, 5428
LIPID STORAGE DISEASES, 6128*
LIVER, 5353*
TOLERANCE
LIVER DISEASE, 5620*
LIVER FUNCTION TESTS, 5616*
RADIOIMMUNOASSAY, 5616*

BILE DUCT
ANOMALY, CONGENITAL
LIVER, 6109*
ATRESIA
DOWNS SYNDROME, 6190
PATHOLOGY, 6211*
PROGNOSIS, 6211*
CALCULI, 6417
CYSTS
DISEASES ASSOCIATED WITH, 6034
ECHOGRAPHY, 6398*
DILATATION
ANOMALY, CONGENITAL, 6415
CHOLELITHIASIS, 6453
HEPATECTOMY, 6453
DISEASE
RADIOLOGY, 5674
FASCIOLIASIS
DRUG TREATMENT, 6557
FIBROSIS
LIVER, 6428
LIVER
ANOMALY, CONGENITAL, 6396*
CYSTS, 6396*
PATHOLOGY, 6168
ULTRASTRUCTURE, 5215*
NEOPLASMS, MALIGNANT
CYTOLOGY, 5631*
DISEASES ASSOCIATED WITH, 6419
SURGERY, 6432, 6435, 6445
OBSTRUCTION
LIPOPROTEINS, 5365*
SHUNT
LIVER, 6109*

BILE DUCT (continued)
SURGERY, 6449, 6451
COMPLICATIONS, 6406*, 6455
SURVIVAL, 6444, 6455
TECHNIQUES, 6450
ULTRASTRUCTURE
ADENOSINE TRIPHOSPHATASE, 5368*

BILE SALTS
SEE BILE ACIDS

BILIARY
ANGIOGRAPHY
BLEEDING, 6434
COMPLICATIONS, 6434
TECHNIQUES, 6210*
ATRESIA
DRUG TREATMENT, 6227
LIPOPROTEINS, 5624*
PATHOLOGY, 6381*
PHENOBARBITAL, 6227
DYSKINESIA
REVIEWS, 6426
FISTULA
SURGERY, 6395*, 6459
HAMARTOMA
SURGERY, 6448
NEOPLASMS, BENIGN
SURGERY, 6448
NEOPLASMS, MALIGNANT
AFLATOXIN, 5483
PRIMATES, 5483
OBSTRUCTION
INFECTION, VIRUS, 5401*
SIMULATION, 5401*
PATHOLOGY
BILIARY DISEASE, 6462
RADIOLOGY, 6392*
SECRETION
DRUG EFFECTS ON, 6178
LIVER DISEASE, 6178
STRICTURE
DILATATION, 6470
SURGERY
ANTIBIOTICS, 6474
COMPLICATIONS, 6458
PANCREATITIS, 6077
SEQUELAE, 6392*

BILIARY DISEASE
ANGIOGRAPHY
TECHNIQUES, 5643, 5645, 5646
BILIARY
PATHOLOGY, 6462
BLOOD
FIBRINOLYSIS, 6188
CHILDREN
CHOLERETIC AGENTS, 6379*
DRUG TREATMENT, 6379*
COMPLICATIONS, 6462
DUODENUM, 5891
DIAGNOSIS, 6185, 6460, 6461
DRUG TREATMENT, 6420
DUODENUM
OBSTRUCTION, 5891
JAUNDICE
REVIEWS, 6429
LIVER
DRUG METABOLISM, 6404*
SCANNING, SCINTILLATION, 5634*

BILIARY DISEASE (continued)
LIVER FUNCTION TESTS
SULFOBROMOPHTHALEIN, 6182
PANCREAS DISEASE
DISEASES ASSOCIATED WITH, 6383*
PEPTIC ULCER
DISEASES ASSOCIATED WITH, 5816
RADIOLOGY, 5678
SCANNING, SCINTILLATION
TECHNIQUES, 5396*
SMALL INTESTINE
MICROORGANISMS, 6411
SURGERY, 6185, 6441
COMPLICATIONS, 6425
TECHNIQUES, 6469
UROGENITAL SYSTEM, 6414
THERAPY, 6457, 6460, 6461

BILIARY TRACT
INFECTION, BACTERIA
ANTIBIOTICS, 6424
DIAGNOSIS, 6424

BILIRUBIN
BINDING
FEEDING, 6215*
GILBERTS DISEASE, 6215*
JAUNDICE, 6214*
GLYCOSIDES
BILE, 6213*
CHROMATOGRAPHY, 6213*
METABOLISM
HODGKINS DISEASE, 6156
LEUKEMIA, 6156
LIVER, 5421, 5439
LIVER DISEASE, 6177
PHOTOTHERAPY, 5461
REVIEWS, 5421, 6177
TEMPERATURE, 5461
SYNTHESIS
CORTICOSTEROIDS, 6216
JAUNDICE, 6216

BIOCHEMISTRY
SMALL INTESTINE
CORTICOSTEROIDS, 5199*

BIOPOTENTIALS
COLON
BILE ACIDS, 5572*
ESOPHAGUS
NERVOUS SYSTEM, 5280
GASTROINTESTINAL
PEPTIDES, 5270*
NERVOUS SYSTEM
CHOLECYSTOKININ, 5603
RECTUM
CARBENOXALONE, 5799*
SMALL INTESTINE
CHOLECYSTECTOMY, 6418
FEEDING, 5259
HORMONE EFFECTS ON, 5259
STOMACH
ASPIRIN, 5258
CARBENOXALONE, 5258, 5799*
CHOLECYSTECTOMY, 6418
PEPTIC ULCER, 5260

BLEEDING
BILIARY
ANGIOGRAPHY, 6434
CECUM
POLYPS, 5995
CIRRHOSIS
ANGIOGRAPHY, 6362
COLON
DIVERTICULUM, 5963
DUODENUM
DIAGNOSIS, 5660
DRUG-INDUCED, 5660
EPIDEMIOLOGY, 5660
SURGERY, 6500
TRACER STUDY, 5633*
GASTROINTESTINAL
ANTACIDS, 6479*
ANTIINFLAMMATORY AGENTS, 6492
ASPIRIN, 6154, 6479*
CIRRHOSIS, 6365, 6375
DRUG-INDUCED, 6479*, 6492
ETIOLOGY, 6501
GASTRECTOMY, 5810
LIVER FUNCTION TESTS, 6365
REVIEWS, 6508
THERAPY, 6501
GASTROINTESTINAL TRACT
TRACER STUDY, 5633*
LIVER
HYPERPLASIA, 6200
TRAUMA, 6129*
MECKELS DIVERTICULUM
SCANNING, SCINTILLATION, 5687
PANCREAS
PSEUDOCYST, 6036, 6066
PANCREATITIS
AMINO ACIDS, 5340*
KIDNEY TRANSPLANTATION, 6071
PROTEASE, 6064
SHOCK
DRUG TREATMENT, 5413*
STOMACH
DIAGNOSIS, 5660
DRUG-INDUCED, 5660
EPIDEMIOLOGY, 5660
EROSIONS, 5794
HYPOTHERMIA, 5794
SURGERY, 6500
TRACER STUDY, 5633*
ULCER, 5797
VOLVULUS, 5793

BLIND LOOP SYNDROME
SMALL INTESTINE
DIVERTICULUM, 5917
PATHOLOGY, 5205*
VITAMIN B12
ABSORPTION, 5902*

BLOOD
ALBUMIN
DIETARY FACTORS, 5456
STARCHES, 5456
SUCROSE, 5456
BILE ACIDS
HYPERBILIRUBINEMIA, 6118*
LIVER DISEASE, 5632*, 6118*, 6124*

BLOOD (continued)
CALCIUM
GASTRECTOMY, 5320
GASTRIN, 5320
PANCREATITIS, 5339*
CARBON DIOXIDE
CIRRHOSIS, 6345*
CHOLESTEROL
CHOLELITHIASIS, 5370*, 6388*
CLOTTING
PANCREATITIS, 6070
COPPER
SCHISTOSOMIASIS, 6003
ESTROGENS
LIVER DISEASE, ALCOHOLIC, 6317*
FETOPROTEIN, ALPHA
LIVER DISEASE, 6117*
FIBRINOLYSIS
BILIARY DISEASE, 6188
LIVER DISEASE, 6188
GASTRIN
ACIDITY, 5306*, 5310*
ALCOHOLISM, 5304*
ANTACIDS, 5838
CALCIUM, 5307*, 5310*
FEEDING, 5313, 5839
MAGNESIUM, 5307*
PEPTIC ULCER, 5804, 5838, 5839
RADIOIMMUNOASSAY, 5685
VAGOTOMY, 5804
ZOLLINGER-ELLISON SYNDROME, 6483*, 6521
GLUCOSE
DUMPING SYNDROME, 5739*
IMMUNOGLOBULINS
CHOLERA, 6513
ECHINOCOCCOSIS, 6550*
PANCREAS DISEASE, 6055*
PANCREATITIS, 6055*
SCHISTOSOMIASIS, 6550*
TOXOPLASMOSIS, 6550*
TRICHINOSIS, 6550*
KININS
DUMPING SYNDROME, 5758
LIPASE
LIVER DISEASE, 6123*, 6147
LIPOPROTEINS
CHOLESTASIS, 6126*
HEPATITIS, 5624*
JAUNDICE, OBSTRUCTIVE, 5365*, 6126*, 6421
LIVER DISEASE, 6126*
MACROAMYLASE
MALABSORPTION, 5908*
METALS
LIVER FUNCTION TESTS, 6140
OBESITY, 6140
MOTILIN
RADIOIMMUNOASSAY, 5273*
OXYGEN
CIRRHOSIS, 6345*
PERFUSION
HEPATITIS, VIRAL, 6239*
LIVER COMA, 6239*
PHOSPHATASE, ALKALINE
DIETARY FACTORS, 5432
DRUG EFFECTS ON, 5541
ISOENZYMES, 6113*
LIVER DISEASE, 6113*

BLOOD (continued)
PHYSICAL PROPERTIES
CIRRHOSIS, 6338*
HEPATITIS, CHRONIC, 6338*
PROTEIN
CROHNS DISEASE, 6581*
LIVER DISEASE, 6137
SCHISTOSOMIASIS, 6569
ULCERATIVE COLITIS, 6581*
PROTHROMBIN
LIVER DISEASE, 6148
SEROTONIN
DUMPING SYNDROME, 5739*
PANCREATITIS, 6074
TRACE ELEMENTS
CHOLECYSTITIS, 6412
TRANSAMINASE
DIETARY FACTORS, 5432
ENVIRONMENTAL FACTORS, 5441
GENETIC FACTORS, 5441
TRIGLYCERIDE
FATTY ACIDS, 5435
LIVER DISEASE, 6123*
VITAMIN A, 5549
ZINC
SCHISTOSOMIASIS, 6003

BLOOD FLOW
SEE CIRCULATION

BLOOD SUBSTITUTES
LIVER
MORPHOLOGY, 5223

BUDD-CHIARI SYNDROME
ETIOLOGY
ORAL CONTRACEPTIVES, 6369
THERAPY, 6369

BURNS
SEE ALSO CHEMICAL BURNS
LIVER
ULTRASTRUCTURE, 5502

CADMIUM
ILEUM
METALS, 5526
LIVER
METALS, 5526
OCCUPATIONAL FACTORS, 6229
PATHOLOGY, 5369*

CALCIFICATION
ABDOMEN
DISEASES ASSOCIATED WITH, 5778
PANCREATITIS, CHRONIC
IMMUNOLOGY, 6053*
LACTOFERRIN, 6050*
PATHOLOGY, 6054*
SECRETION, 6054*
STOMACH, 6059
STOMACH
NEOPLASMS, MALIGNANT, 5778

CALCITONIN
STOMACH
ACID SECRETION, 5769
MOTILITY, 5769

CALCIUM
ABSORPTION
DIETARY FACTORS, 5263
SMALL INTESTINE, 5263
AMYLASE
SECRETION, 5350
BLOOD
GASTRECTOMY, 5320
GASTRIN, 5307*, 5310*, 5320
PANCREATITIS, 5339*
COLON
MOTILITY, 5268*
DEFICIENCY
ABSORPTION, 5263
DUODENUM
PANCREATITIS, CHRONIC, 6051*
PARATHYROID GLAND, 6051*
ILEUM
MOTILITY, 5268*, 5283
LIVER
PHAGOCYTOSIS, 5391*
PANCREAS
SECRETION, 5337*
PHOSPHOLIPID
METABOLISM, 5350
STOMACH
ACID SECRETION, 5307*, 5310*

CALCULI
SEE ALSO GALLSTONE, CHOLELITHIASIS, CHOLEDOCHOLITHIASIS
BILE DUCT, 6417
PANCREAS
AMYLASE, 6027*
BILE, 6027*
GLUTAMYL TRANSPEPTIDASE, GAMMA, 6027*
SECRETION, 6027*

CANCER
SEE NEOPLASMS, MALIGNANT

CARBENOXALONE
PEPTIC ULCER
DRUG TREATMENT, 5799*
RECTUM
BIOPOTENTIALS, 5799*
STOMACH
ABSORPTION, 5258
BIOPOTENTIALS, 5258, 5799*

CARBOHYDRATE
SEE ALSO SPECIFIC SUGARS
INTOLERANCE
COMPLICATIONS, 6517
DIARRHEA, 6517
LIVER DISEASE
DIETARY FACTORS, 6084*
LIVER FUNCTION TESTS
DIETARY FACTORS, 6084*
METABOLISM
CORTICOSTEROIDS, 5448
ISOTHIOCYANATES, 5448
LIVER, 5448, 5496
LIVER INJURY, 5382*
SHOCK, 5496

CARBON DIOXIDE
BLOOD
CIRRHOSIS, 6345*

CARBON DISULFIDE
METABOLISM
LIVER, 5491

CARBON TETRACHLORIDE
BILE
EXCRETION, 5500
BILE ACIDS
METABOLISM, 5480
LIVER
AMINO ACIDS, 5419*
LIPIDS, 5516
PEROXIDATION, 5419*
SIALIC ACID, 5419*
LIVER INJURY, 5535
COENZYMES, 5382*
CYTOCHROMES, 5485
DETERGENTS, 5436
DRUG TREATMENT, 5548
PHOSPHOLIPID, 5419*
THERAPY, 6225*

CARCINOGENS
LIVER
MITOCHONDRIA, 5497
NUCLEIC ACIDS, 5458
PATHOLOGY, 5527
RIBOSOMES, 5458
METABOLISM
LIVER, 5527

CARCINOIDS
APPENDIX
SURGERY, 5970
GASTROINTESTINAL
DISEASES ASSOCIATED WITH, 6487
NEOPLASMS, MALIGNANT, 6487
SMALL INTESTINE
CROHNS DISEASE, 5934*

CATHARTICS
SEE LAXATIVES

CECUM
SEE ALSO LARGE INTESTINE
COLON
NEOPLASMS, MALIGNANT, 5998
LACTASE
DIETARY FACTORS, 5573*
POLYPS
BLEEDING, 5995
PATHOLOGY, 5995
RUPTURE
NEOPLASMS, MALIGNANT, 5998
VOLVULUS
SURGERY, 5991

CELIAC DISEASE
DIAGNOSIS, 5912
DISEASES ASSOCIATED WITH
NEOPLASMS, MALIGNANT, 5921
UROGENITAL SYSTEM, 5923
DUODENUM
PATHOLOGY, 5909*
IMMUNOLOGY
LEUKOCYTE, 5901*
JEJUNUM
ATROPHY, 5921

CELIAC DISEASE (continued)
POTASSIUM
ABSORPTION, 5910*
PREGNANCY
DIET, 5906*
SMALL INTESTINE
ENZYMES, 5222
REGENERATION, 5913
TISSUE CULTURE, 5907*
ULTRASTRUCTURE, 5222
THERAPY
DIET, 5909*

CERULEIN
PANCREAS FUNCTION TESTS, 5657
PANCREATITIS
DIAGNOSIS, 5657

CHELATING AGENTS
HEPATITIS, CHRONIC
DRUG TREATMENT, 6293*, 6294*
SMALL INTESTINE
MORPHOLOGY, 5226
PERMEABILITY, 5226

CHEMICAL BURNS
STOMACH
ENDOSCOPY, 5754

CHEMICAL COMPOSITION
BILE
CHOLECYSTECTOMY, 6376*
GLYCOPROTEINS, 5481
FECES
GASTROENTEROSTOMY, 5814
PEPTIC ULCER, 5814
LIVER
BILE ACIDS, 5552
LIPIDS, 5493
PHOSPHOLIPID, 5540

CHEMOTAXIS
LEUKOCYTES
LIVER DISEASE, ALCOHOLIC, 6316*

CHEMOTHERAPY
DUODENUM
NEOPLASMS, MALIGNANT, 6484*
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 5936*
LIVER
NEOPLASMS, MALIGNANT, 6088*, 6196, 6199, 6206
STOMACH
NEOPLASMS, MALIGNANT, 6484*

CHENODEOXYCHOLIC ACID
BILE
CHOLESTEROL, 5366*
LIPIDS, 5366*
BILE ACIDS
METABOLISM, 6378*, 6386*, 6393*
CHOLELITHIASIS
DRUG TREATMENT, 6378*, 6385*, 6386*, 6433, 6456
TOXICITY
PRIMATES, 5386*
TRIGLYCERIDE
METABOLISM, 6380*

CHILDREN
SEE ALSO NEONATE, AGE FACTORS
BILIARY DISEASE
CHOLERETIC AGENTS, 6379*
DRUG TREATMENT, 6379*
CHOLANGITIS
DIAGNOSIS, 5653
CHOLECYSTITIS
DIAGNOSIS, 5653
DISEASES ASSOCIATED WITH, 6473
CHOLELITHIASIS, 6384*
FAMILIAL FACTORS, 6439
CIRRHOSIS
ANTIGEN, AUSTRALIA, 6246*
DISEASES ASSOCIATED WITH, 6246*
GENETICS, 6333*
HEPATITIS, VIRAL, 6246*
THERAPY, 6296
CROHNS DISEASE
DIAGNOSIS, 6579*
DEFECATION
RADIOLOGY, 5638*
DIARRHEA
DRUG TREATMENT, 6493
DUODENITIS
PATHOLOGY, 5874
GASTRODUODENITIS
DIAGNOSIS, 6488
IMMUNOGLOBULINS, 6488
GASTROINTESTINAL
FOREIGN BODIES, 6476*
HEPATITIS, CHRONIC
IMMUNOSUPPRESSION, 6291*
STEROID, 6291*
THERAPY, 6296
HEPATITIS, SERUM
ANTIGEN, AUSTRALIA, 6260
HEPATITIS, VIRAL, 6252
ANTIGEN, AUSTRALIA, 6260
DIAGNOSIS, 6268
DRUG TREATMENT, 6259
HYDROCARBONS, 6259
IMMUNOLOGY, 6284
SEQUELAE, 6270
TRACE ELEMENTS, 6234*
LARGE INTESTINE
NEOPLASMS, 5870
PSEUDOTUMOR, 5870
LIVER
NEOPLASMS, MALIGNANT, 6149
TRAUMA, 6193
LIVER DISEASE
FETOPROTEIN, ALPHA, 6189
REVIEWS, 6208
MECKELS DIVERTICULUM, 5871
MEGACOLON
ANOMALY, CONGENITAL, 5953
PANCREAS
CIRCULATION, 5217
PARASITES AND PARASITIC DISEASE
EPIDEMIOLOGY, 6572
PEPTIC ULCER
EPIDEMIOLOGY, 5811
SALMONELLOSIS
TRANSMISSION, 6538
SMALL INTESTINE
NEOPLASMS, 5870
PSEUDOTUMOR, 5870

CHILDREN (continued)
VITAMIN B12
MALABSORPTION, 5918

CHOLANGIOGRAPHY
SEE ANGIOGRAPHY

CHOLANGITIS
ANTIBIOTICS
TECHNIQUES, 6443
DIAGNOSIS
CHILDREN, 5653

CHOLECYSTECTOMY
BILE
CHEMICAL COMPOSITION, 6376*
BILE ACIDS
BINDING, 6405*
CHOLECYSTITIS, 6471
DRAINAGE
DRUG ABSORPTION, 6401*
GALLBLADDER DISEASE, 6389*
SMALL INTESTINE
BIOPOTENTIALS, 6418
STOMACH
BIOPOTENTIALS, 6418

CHOLECYSTITIS
ANTIBIOTICS
TECHNIQUES, 6443
BILE
BACTERIA, 6465
BILE ACIDS, 6454
BLOOD
TRACE ELEMENTS, 6412
CHILDREN
DISEASES ASSOCIATED WITH, 6473
CHOLECYSTECTOMY, 6471
COMPLICATIONS
FISTULA, 6438
DIAGNOSIS
CHILDREN, 5653
DUODENUM
PRESSURE STUDIES, 6447
EMPHYSEMA, 6332*
GALLBLADDER
ULTRASTRUCTURE, 6394*
STOMACH
SECRETION, 6489
SURGERY, 6437

CHOLECYSTOKININ
APPETITE
HORMONE CONTROL, 5603
DUODENUM
ACIDITY, 5574*
NERVOUS SYSTEM
BIOPOTENTIALS, 5603
PANCREAS
SECRETION, 5574*
SMALL INTESTINE
ENZYMES, 5583
STOMACH
GASTRIN, 5321
MOTILITY, 5271*

CHOLEDOCHOLITHIASIS
DIAGNOSIS, 6463

CHOLEDOCHOLITHIASIS (continued)
DRUG TREATMENT
CHOLIC ACID, 6402*
PAPILLA OF VATER
SURGERY, 6400*
PATHOLOGY, 6446
RADIOLOGY, 6446
SURGERY, 6463
THERAPY
TECHNIQUES, 6402*

CHOLELITHIASIS
BILE ACIDS
METABOLISM, 5403*, 6386*
BILE DUCT
DILATATION, 6453
BLOOD
CHOLESTEROL, 5370*, 6388*
CHILDREN, 6384*
FAMILIAL FACTORS, 6439
CHOLESTEROL
SYNTHESIS, 6388*
DISEASES ASSOCIATED WITH
PANCREATITIS, CHRONIC, 6377*, 6391*
DRUG-INDUCED
STEROID, 5544
DRUG TREATMENT
CHENODEOXYCHOLIC ACID, 6378*, 6385*, 6386*, 6433, 6456
ETIOLOGY
BILE ACIDS, 6413
GALLBLADDER
PATHOLOGY, 5230
ULTRASTRUCTURE, 6394*
OBESITY
SHUNT, INTESTINAL, 6116*
PANCREAS
SECRETION, 6391*
PANCREAS FUNCTION TESTS
SECRETIN, 6391*
PANCREATITIS, CHRONIC
ETIOLOGY, 6377*
PROGNOSIS
AGE FACTORS, 6422
SHUNT, INTESTINAL
COMPLICATIONS, 6403*
SEQUELAE, 6390*
SIMULATION
PRIMATES, 5370*
THERAPY
AGE FACTORS, 6468
BILE ACIDS, 6413
VITAMIN C
DEFICIENCY, 5544

CHOLERA
BLOOD
IMMUNOGLOBULINS, 6513
CARRIER STATE
IMMUNOLOGY, 6513
DRUG TREATMENT
ANTIBIOTICS, 6539
ANTIDIARRHEALS, 6539
IMMUNOLOGY
SMALL INTESTINE, 5602
SMALL INTESTINE
IMMUNOLOGY, 5601
PERISTALSIS, 5601

CHOLERA (continued)
SODIUM
TRANSPORT, 5256
STOMACH
ACID SECRETION, 5601
THERAPY
SUCROSE, 6478*
TOXIN
SODIUM, 5256
VACCINE
IMMUNITY, 6527

CHOLERESIS
DRUG-INDUCED
ASPIRIN, 5431

CHOLERETIC AGENTS
BILIARY DISEASE
CHILDREN, 6379*
CONTRAST MEDIA
EXCRETION, 5362*
GASTROINTESTINAL
MOTILITY, 5289
SECRETION, 5289

CHOLESTASIS
BILE
BILE ACIDS, 6387*
GLUTAMYL TRANSPEPTIDASE, GAMMA, 6387*
PHOSPHATASE, ALKALINE, 6387*
BILE ACIDS
METABOLISM, 6120*
BLOOD
LIPOPROTEINS, 6126*
DIAGNOSIS
LIVER FUNCTION TESTS, 6126*
ERYTHROCYTE
LIPIDS, 6114*
LIPOPROTEINS
DIAGNOSIS, 6181
LIVER
ETIOLOGY, 5408*, 6135
HEPATITIS, VIRAL, 6240*
ISOTHIOCYANATES, 5448
PREGNANCY, 6108*
REVIEWS, 6427
SURGERY, 6135
ULTRASTRUCTURE, 5408*
PREGNANCY
REVIEWS, 6159

CHOLESTEROL
BILE
CHENODEOXYCHOLIC ACID, 5366*
BLOOD
CHOLELITHIASIS, 5370*, 6388*
EXCRETION
BILE, 5424
BILE ACIDS, 5424
FATTY LIVER, 5536
DIETARY FACTORS, 5473
JEJUNUM
TRANSPORT, 5248*
LARGE INTESTINE
BACTERIA, 5928*
LIVER
HEPATITIS, VIRAL, 6240*
PATHOLOGY, 5536

CHOLESTEROL (continued)
METABOLISM
CIRRHOSIS, 5473
LAXATIVES, 5543
LIVER, 5379*, 5543, 5614
OPSONIN, 5614
THYROID, 5379*
SYNTHESIS
CHOLELITHIASIS, 6388*
COENZYMES, 5471
DIETARY FACTORS, 5529
DRUG EFFECTS ON, 6194
LIPOPROTEINS, 5358*
LIVER, 5358*, 5471, 5529
LIVER DISEASE, 6194
SMALL INTESTINE, 5579

CHOLIC ACID
CHOLEDOCHOLITHIASIS
DRUG TREATMENT, 6402*

CHOLINE
ABSORPTION
SMALL INTESTINE, 5238*
DEFICIENCY
PANCREATITIS, 5340*

CHOLINERGIC AGENTS
PANCREAS
SECRETION, 5337*
SMALL INTESTINE
CYCLIC GUANOSINE MONOPHOSPHATE, 5575*

CHOLINESTERASE
STOMACH
MOTILITY, 5272*

CHYME
NITROGEN
FEEDING, 5290
SMALL INTESTINE, 5290
STOMACH, 5290

CHYMOTRYPSIN
INTESTINE
ABSORPTION, 5342*
SECRETION
PANCREAS, 5341*

CIRCADIAN RHYTHM
BILE
BILE ACIDS, 6387*
PHOSPHATASE, ALKALINE, 6387*
BILE ACIDS
SYNTHESIS, 5428
SMALL INTESTINE
ENZYMES, 5561*

CIRCULATION
ANORECTUM
MORPHOLOGY, 5608
SURGERY, 5608
CIRRHOSIS, 6341*
ASCITES, 6349*
HEPATITIS, VIRAL
DISEASES ASSOCIATED WITH, 6287
INTESTINE
ECHOGRAPHY, 5585

CIRCULATION (continued)
JEJUNUM
TECHNIQUES, 5599*
KIDNEY
HEPATORENAL SYNDROME, 6346*
LIVER
CIRRHOSIS, 6350
GLUCOSE, 5399*
JAUNDICE, OBSTRUCTIVE, 6416
LIVER DISEASE, 5682
LIVER FUNCTION TESTS, 5420
MORPHOLOGY, 5210*
PATHOLOGY, 6169
PERFUSION, 5438
PORTAL HYPERTENSION, 6343*
PRIMATES, 5210*
SCANNING, SCINTILLATION, 6350
SECRETIN, 5445
SEPSIS, 5404*
STARVATION, 5404*
SURGERY, 6416
VASOPRESSIN, 5393*, 5415*, 5533
LIVER DISEASE
DISEASES ASSOCIATED WITH, 6130
PANCREAS
CHILDREN, 5217
GASTROENTEROSTOMY, 6030*
PANCREATITIS
DISEASES ASSOCIATED WITH, 6070
PEPTIC ULCER, 5830
DISEASES ASSOCIATED WITH, 5837
PORTAL HYPERTENSION, 6341*
VASOPRESSIN, 6348*
SMALL INTESTINE
TRANSPLANTATION, 5711
STOMACH
ADRENERGIC AGENTS, 5597*
ALCOHOL, 5590*
ASPIRIN, 5590*
BILE ACIDS, 5590*
CORTICOSTEROIDS, 5317
DRUG EFFECTS ON, 5300*
NEOPLASMS, MALIGNANT, 5757
PERMEABILITY, 5265
VAGOTOMY, 5300*, 5326
VASOCONSTRICTORS, 5597*
XYLOSE
ABSORPTION, 5925

CIRRHOSIS
ACIDOSIS, 6354
ALCOHOLISM
IMMUNOLOGY, 6328
REVIEWS, 6326
ALKALOSIS, 6354
ANEMIA
DISEASES ASSOCIATED WITH, 6371
ANGIOGRAPHY, 6363
ANTIGEN, AUSTRALIA, 6236*, 6245*
IMMUNOLOGY, 6273
ANTITRYPSIN, ALPHA
DEFICIENCY, 6333*
ASCITES
PORTAL HYPERTENSION, 6351
REVIEWS, 6359
THERAPY, 6351
BILE ACIDS
METABOLISM, 6105*

CIRRHOSIS (continued)
BLEEDING
ANGIOGRAPHY, 6362
BLOOD
PHYSICAL PROPERTIES, 6358*
CARBON DIOXIDE
BLOOD, 6345*
CHILDREN
ANTIGEN, AUSTRALIA, 6246*
DISEASES ASSOCIATED WITH, 6246*
GENETICS, 6333*
HEPATITIS, VIRAL, 6246*
THERAPY, 6296
CHOLESTEROL
METABOLISM, 5473
CIRCULATION, 6341*
ASCITES, 6349*
CORTICOSTEROIDS
METABOLISM, 6097*, 6127*
DIET, 6353
DISEASES ASSOCIATED WITH
NERVOUS SYSTEM, 6370
RESPIRATORY SYSTEM, 6153
DRUG TREATMENT, 6299, 6371
CORTICOSTEROIDS, 6289*
GLUTATHIONE, 6352
IMMUNOSUPPRESSION, 6289*
NERVOUS SYSTEM, 6335*
VITAMIN B12, 6352
ERYTHROCYTE
LIPIDS, 6114*
ESOPHAGUS
VARICES, 6336*
ETIOLOGY
ALCOHOL, 6318*
DIETARY FACTORS, 6318*
FAMILIAL FACTORS, 6318*
MALNUTRITION, 6353
GASTROINTESTINAL
BLEEDING, 6365, 6375
GLUCOSE
METABOLISM, 6335*
HEPATITIS, CHRONIC
COMPLICATIONS, 6297
HEPATITIS, VIRAL
SEQUELAE, 6265
IMMUNOGLOBULINS, 6245*
IMMUNOLOGY, 6314*
LAPAROSCOPY, 5694
LIPIDS
METABOLISM, 6301
LIVER
CIRCULATION, 6350
COLLAGEN, 6119*
CONNECTIVE TISSUE, 6119*
DRUG METABOLISM, 6078*
METASTASES, 6356
NEOPLASMS, 6106*
NEOPLASMS, MALIGNANT, 6091*
LIVER FUNCTION TESTS, 6337*
MALNUTRITION
AMINO ACIDS, 6366
DRUG TREATMENT, 6366
NERVOUS SYSTEM, 6337*
NUCLEIC ACIDS
SYNTHESIS, 6244*, 6292*
OBESITY
SHUNT, INTESTINAL, 6111*

CIRRHOSIS (continued)
OXYGEN
BLOOD, 6345*
PORTAL HYPERTENSION
SURGERY, 6372
SURVIVAL, 6372
PROTEIN
DEFICIENCY, 5473
RESPIRATORY SYSTEM
DRUG EFFECTS ON, 6345*
REVIEWS, 6327, 6361
RISK FACTORS
ALCOHOL, 6339*
SCANNING, SCINTILLATION, 6368
STEROID
URINE, 6355
STOMACH
TELANGIECTASIA, 6358
SULFOBROMOPHTHALEIN
KINETICS, 6334*
SURGERY
ANGIOGRAPHY, 6362
THERAPY, 6373, 6374
REVIEWS, 6187, 6300
WATER
ABSORPTION, 6340*
EXCRETION, 6340*

CIRRHOSIS, BILIARY
AUTOIMMUNITY
REVIEWS, 6101*
COPPER
METABOLISM, 6344*
PIGMENTATION, 6344*
DISEASES ASSOCIATED WITH, 6344*
ETIOLOGY
AUTOIMMUNITY, 6298

CLOTTING
BLOOD
PANCREATITIS, 6070
LIVER INJURY
GALACTOSAMINE, 5446
PANCREAS
NEOPLASMS, MALIGNANT, 6037

COBALT
HEME
METABOLISM, 5356*

COENZYMES
ALCOHOL
METABOLISM, 6315*
CHOLESTEROL
SYNTHESIS, 5471
LIVER
KETOGENESIS, 5471
LIVER INJURY
CARBON TETRACHLORIDE, 5382*
PHOSPHOLIPID
SYNTHESIS, 5238*

COLITIS
SEE ALSO ULCERATIVE COLITIS
DRUG-INDUCED
ANTIBIOTICS, 5930*, 6008
ESOPHAGUS
SURGERY, 5985

COLITIS (continued)
GLYCOGEN STORAGE DISEASE
DISEASES ASSOCIATED WITH, 6204
ISCHEMIA
ESOPHAGUS, 5985
PATHOLOGY, 5987
RADIOLOGY, 5987
STOMACH
SECRETION, 6489

COLITIS, GRANULOMATOUS
ENDOSCOPY, 5622*

COLLAGEN
LIVER
CIRRHOSIS, 6119*
FIBROSIS, 6119*

COLON
SEE ALSO LARGE INTESTINE
AMEBIASIS
DISEASES ASSOCIATED WITH, 6552
ANOMALY, CONGENITAL, 5962
GASTRECTOMY, 5954
BIOPOTENTIALS
BILE ACIDS, 5572*
CYSTS
RADIOLOGY, 5993
DISEASES
EPIDEMIOLOGY, 5984
DIVERTICULUM
BLEEDING, 5963
COMPLICATIONS, 5959
SURGERY, 5959, 5963
ENDOSCOPY
COMPLICATIONS, 6006
REVIEWS, 5698
FISTULA
STOMACH DISEASE, 5790
HERNIA
GASTRECTOMY, 5954
HODGKINS DISEASE, 6010
LYMPHAGIECTASIA, 5951
MORPHOLOGY
BILE ACIDS, 5245*
MOTILITY
BILE ACIDS, 5245*
CALCIUM, 5268*
ELECTROLYTE, 5268*
NERVOUS CONTROL, 5276*
MUCOPOLYSACCHARIDE
ENZYMES, 5211*
HYDROLYSIS, 5211*
NECROSIS
AMEBIASIS, 5992
ETIOLOGY, 5972
SURGERY, 5972
NEOPLASMS
PARASITES AND PARASITIC DISEASE, 6554
NEOPLASMS, MALIGNANT, 5868*
ANTIGEN, CARCINOEMBRYONIC, 5935*
CECUM, 5998
DIAGNOSIS, 5981
ENDOSCOPY, 5692
ISCHEMIA, 5965
MELENA, 5975
PATHOLOGY, 5978
PERFORATION, 5975

COLON (continued)
NEOPLASMS, MALIGNANT (continued)
RADIOLOGY, 5932*
SURGERY, 5974, 5981
SURVIVAL, 6000
PERFORATION
AMEBIASIS, 5992
SCHISTOSOMIASIS, 5983
SURGERY, 5955, 6002
PNEUMATOSIS, 5945
POLYPS
ENDOSCOPY, 5980
PATHOLOGY, 5947
PRECANCER, 5943, 5947
REVIEWS, 6001
SURGERY, 5946, 5980
PRECANCER
REVIEWS, 5956
PSEUDOTUMOR
AMEBIASIS, 6007
RADIOLOGY
GLYCOGEN STORAGE DISEASE, 6204
TECHNIQUES, 5658, 5679
SECRETION
BILE ACIDS, 5572*
STENOSIS
PANCREATITIS, 6061
STOMACH
FISTULA, 5768
SURGERY
SEQUELAE, 5952
TRANSPORT
ADENYL CYCLASE, 5204*
ULTRASTRUCTURE
ADENYL CYCLASE, 5204*
PEPTIDES, 5567*

COMA
LIVER
PREVENTION, 6342*
STEROID, 6342*

COMPLEMENT
HEPATITIS, CHRONIC
ANTIGEN, AUSTRALIA, 6290*

CONNECTIVE TISSUE
LIVER
CIRRHOSIS, 6119*
FIBROSIS, 6119*

CONSTIPATION
DEFECATION
RADIOLOGY, 5638*
INTESTINE
MOTILITY, 6482*
PSYCHOLOGICAL FACTORS, 5994

CONTRAST MEDIA
EXCRETION
CHOLERETIC AGENTS, 5362*
LIVER, 5362*, 5683
LIVER
NEOPLASMS, MALIGNANT, 6201

COPPER
BLOOD
SCHISTOSOMIASIS, 6003
METABOLISM
CIRRHOSIS, BILIARY, 6344*

COPPER (continued)
METABOLISM (continued)
JAUNDICE, OBSTRUCTIVE, 5394*
LIVER, 5394*
PIGMENTATION
CIRRHOSIS, BILIARY, 6344*

CORTICOSTEROIDS
BILIRUBIN
SYNTHESIS, 6216
CARBOHYDRATE
METABOLISM, 5448
CIRRHOSIS
DRUG TREATMENT, 6289*
HEPATITIS, CHRONIC
ANTIGEN, AUSTRALIA, 6306
DRUG TREATMENT, 6289*, 6294*, 6338*
LIVER
RUPTURE, 6198
METABOLISM
CIRRHOSIS, 6097*, 6127*
FATTY LIVER, 6127*
HEPATITIS, CHRONIC, 6097*, 6127*
LIVER DISEASE, 6097*
LIVER DISEASE, ALCOHOLIC, 6097*
NUCLEIC ACIDS
SYNTHESIS, 5490
SMALL INTESTINE
BIOCHEMISTRY, 5199*
MORPHOLOGY, 5199*
STOMACH
CIRCULATION, 5517
ULCER, 5755
STOMACH DISEASE
DRUG-INDUCED, 5776

CROHNS DISEASE
BLOOD
PROTEIN, 6581*
DIAGNOSIS
ADOLESCENTS, 6579*
CHILDREN, 6579*
DISEASES ASSOCIATED WITH
APPENDICITIS, 6577*
DRUG TREATMENT, 6578*
RECURRENCE, 6588
FAMILIAL FACTORS, 6584
IMMUNOLOGY, 6580*
LARGE INTESTINE
THERAPY, 6505
LIVER
PATHOLOGY, 6587
MOUTH, 6582*
PATHOLOGY
JEJUNUM, 6583
PRECANCER
TRANSFORMATION, 6591
SALIVARY GLANDS
SECRETION, 6582*
SMALL INTESTINE
CARCINOIDS, 5934*
FISTULA, 6593
SURGERY, 6590
RECURRENCE, 6588
THERAPY
DIET, 6589

CRYOSURGERY
LIVER
GLUCURONIDASE, BETA, 5512
LYSOSOMES, 5512

CYCLIC ADENOSINE MONOPHOSPHATE
FATTY ACIDS
SYNTHESIS, 5546
LIVER
ENDOTOXIN, 5507
LEAD, 5507
NUCLEIC ACIDS
SYNTHESIS, 5555
PROTEIN
SYNTHESIS, 5495
STOMACH
ALCOHOL, 5302*

CYCLIC GUANOSINE MONOPHOSPHATE
SMALL INTESTINE
CHOLINERGIC AGENTS, 5575*
HORMONE EFFECTS ON, 5575*
PROSTAGLANDIN, 5575*

CYSTS
BILE DUCT
DISEASES ASSOCIATED WITH, 6034
ECHOGRAPHY, 6398*
COLON
RADIOLOGY, 5993
ECHINOCOCCOSIS
LIVER, 6570
RUPTURE, 6570
LIVER
ANOMALY, CONGENITAL, 6121*
BILE DUCT, 6396*
DISEASES ASSOCIATED WITH, 6419
ECHOGRAPHY, 5636*
HEPATECTOMY, 6152
PANCREAS
ANGIOGRAPHY, 6040
SURGERY, 6032

CYTOCHROMES
BILE ACIDS
METABOLISM, 6120*
LIVER
ALCOHOL, 5406*
LIVER INJURY
AGE FACTORS, 5485
CARBON TETRACHLORIDE, 5485

CYTOLOGY
BILE DUCT
NEOPLASMS, MALIGNANT, 5631*
PANCREAS
NEOPLASMS, MALIGNANT, 5631*
TRANSPLANTATION, 5346

CYTOTOXICITY
PANCREAS
ALKALOIDS, 5208*

DEATH
SEE ALSO SURVIVAL

DEFECATION
ANORECTUM
ANOMALY, CONGENITAL, 5638*
RADIOLOGY
CHILDREN, 5638*
CONSTIPATION, 5638*
HIRSCHSPRUNGS DISEASE, 5638*

DEHYDROGENASE
ALCOHOL
LIVER, 5528
ALDEHYDES
LIVER, 5539

DERMATITIS HERPETIFORMIS
IMMUNOLOGY
LEUKOCYTE, 5901*
SMALL INTESTINE
PATHOLOGY, 5905*

DETERGENTS
LIVER INJURY
CARBON TETRACHLORIDE, 5436

DETOXIFICATION
BILE ACIDS, 6393*

DIARRHEA
CARBOHYDRATE
INTOLERANCE, 6517
CHILDREN
DRUG TREATMENT, 6493
DRUG TREATMENT, 5893
ESCHERICHIA COLI
SEROLOGY, 6486*
ETIOLOGY
REVIEWS, 6509
ILEOSTOMY
PREVENTION, 5861*
ILEUM
SURGERY, 5893
INTESTINE
MOTILITY, 6482*
JEJUNUM
ULTRASTRUCTURE, 6524
LACTOSE
INTOLERANCE, 6491
NEONATE
DRUG TREATMENT, 6490
SHIGELLOSIS
ETIOLOGY, 6537
TOXIN, 6537
THERAPY
SUCROSE, 6478*
VAGOTOMY
THERAPY, 5859

DIET
CELIAC DISEASE
PREGNANCY, 5906*
THERAPY, 5909*
CIRRHOSIS, 6353
CROHNS DISEASE
THERAPY, 6589
GASTROENTERITIS
THERAPY, 6507
LIVER DISEASE
THERAPY, 6165

DIET (continued)
PORTACAVAL SHUNT
COMPLICATIONS, 6353
ULCERATIVE COLITIS
ILEOSTOMY, 6022

DIETARY FACTORS
ALLERGY
GASTROENTERITIS, 6480*
AMINO ACIDS
METABOLISM, 5468
BILE ACIDS
METABOLISM, 5403*
BLOOD
ALBUMIN, 5456
PHOSPHATASE, ALKALINE, 5432
TRANSAMINASE, 5432
CALCIUM
ABSORPTION, 5263
CECUM
LACTASE, 5573*
CHOLESTEROL
SYNTHESIS, 5529
CIRRHOSIS
ETIOLOGY, 6318*
FATTY LIVER
CHOLESTEROL, 5473
INTESTINE
DISACCHARIDASE, 6526
LACTOSE TOLERANCE, 5924
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 5927*
LIPIDS
METABOLISM, 5383*
SYNTHESIS, 5569*
LIVER
FATTY ACIDS, 5433
LIPIDS, 5433
UREA, 5354*
LIVER DISEASE
CARBOHYDRATE, 6084*
LIVER FUNCTION TESTS
CARBOHYDRATE, 6084*
SMALL INTESTINE
LACTASE, 5573*

DISACCHARIDASE
SEE ALSO LACTASE, SUCRASE
DEFICIENCY
SMALL INTESTINE, 5919
INTESTINE
DIETARY FACTORS, 6526
MALABSORPTION, 6526
SMALL INTESTINE
PHYSICAL PROPERTIES, 5582

DISACCHARIDE
TOLERANCE TESTS
MALABSORPTION, 5670

DIVERTICULITIS
LARGE INTESTINE
REVIEWS, 5968
THERAPY, 6505

DIVERTICULUM
COLON
BLEEDING, 5963

DIVERTICULUM (continued)
COLON (continued)
COMPLICATIONS, 5959
SURGERY, 5959, 5963
DUODENUM, 5872
RADIOLOGY, 5863*
SURGERY, 5863*
GALLBLADDER, 6430
LARGE INTESTINE
RADIOLOGY, 6005
REVIEWS, 5968
SMALL INTESTINE
BLIND LOOP SYNDROME, 5917
MALABSORPTION, 5917
OBSTRUCTION, 5869
PEPTIC ULCER, 5875
STOMACH
DISEASES ASSOCIATED WITH, 5835
PEPTIC ULCER, 5875
ULCER
DUODENUM, 5886

DOWNS SYNDROME
BILE DUCT
ATRESIA, 6190

DRAINAGE
CHOLECYSTECTOMY
DRUG ABSORPTION, 6401*
LIVER
ABSCESS, 5627*
HEMATOMA, 6203
TRAUMA, 6203
PEPTIC ULCER
VAGOTOMY, 5850

DRUG ABSORPTION
CHOLECYSTECTOMY
DRAINAGE, 6401*
SMALL INTESTINE
DYES, 5262

DRUG ADDICTION
LIVER INJURY, 6228

DRUG-INDUCED
CHOLELITHIASIS
STEROID, 5544
CHOLERESIS
ASPIRIN, 5431
COLITIS
ANTIBIOTICS, 5930*, 6008
DUODENUM
BLEEDING, 5660
ENTEROCOLITIS
ANTIBIOTICS, 6534
FATTY LIVER
AMINO ACIDS, 5474
ANTIBIOTICS, 5559
GASTROINTESTINAL
BLEEDING, 6479*, 6492
HEPATITIS
AUTOIMMUNITY, 6101*
HEPATITIS, NONVIRAL
ANESTHETICS, 6226, 6230
ANTIBACTERIALS, 6217*
HYDROCARBONS, HALOGENATED, 6226
HYPERBILIRUBINEMIA
HORMONE, 6212*

DRUG-INDUCED (continued)
ILEUM
MOTILITY, 5283
JAUNDICE
NEONATE, 6212*
LIVER INJURY
ANESTHETICS, 5518, 6232
ANTIARRHYTHMIC AGENTS, 6219*
ANTIBACTERIALS, 6218*
ANTIBIOTICS, 6223*
ANTIINFLAMMATORY AGENTS, 6222*
ANTIMETABOLITES, 5294
GALACTOSAMINE, 5385*
LAXATIVES, 6272
ORAL CONTRACEPTIVES, 6221*
ULTRASTRUCTURE, 5518
PANCREATITIS
PATHOLOGY, 5229
STOMACH
BLEEDING, 5660
STOMACH DISEASE
CORTICOSTEROIDS, 5776

DRUG METABOLISM
LIVER
ALCOHOL, 5558
ANALGESICS, 5451
ANILINE, 5451
ANTIBACTERIALS, 6218*
ANTICOAGULANTS, 5453
ANTIDIABETIC AGENTS, 6150
ANTINEOPLASTIC AGENTS, 5295
BILIARY DISEASE, 6404*
CIRRHOSIS, 6078*
ENZYMES, 5553
HEPATITIS, CHRONIC, 6310
HYDROCARBONS, 5553
HYDROCARBONS, POLYCYCLIC, 5558, 6150
INSECTICIDES, 5554
LIVER COMA, 6078*
LIVER DISEASE, 6150
PRIMATES, 5295
REVIEWS, 5440
SEX FACTORS, 5558
LIVER COMA
PROGNOSIS, 6078*
LIVER DISEASE, ALCOHOLIC, 6330
SALIVARY GLANDS
ANTINEOPLASTIC AGENTS, 5295
PRIMATES, 5295
SMALL INTESTINE
ANTINEOPLASTIC AGENTS, 5295
PRIMATES, 5295

DRUG TOXICITY
LIVER, 5548

DRUG TREATMENT
BILE DUCT
FASCIOLIASIS, 6557
BILIARY
ATRESIA, 6227
BILIARY DISEASE, 6420
CHILDREN, 6379*
BLEEDING
SHOCK, 5413*
CHOLEDOCHOLITHIASIS
CHOLIC ACID, 6402*

DRUG TREATMENT (continued)
CHOLELITHIASIS
CHENODEOXYCHOLIC ACID, 6378*, 6385*, 6386*, 6433, 6456
CHOLERA
ANTIBIOTICS, 6539
ANTIDIARRHEALS, 6539
CIRRHOSIS, 6299, 6371
CORTICOSTEROIDS, 6289*
GLUTATHIONE, 6352
IMMUNOSUPPRESSION, 6289*
MALNUTRITION, 6366
NERVOUS SYSTEM, 6335*
VITAMIN B12, 6352
CROHNS DISEASE, 6578*
RECURRENCE, 6588
DIARRHEA, 5893
CHILDREN, 6493
NEONATE, 6490
DYSPEPSIA, 5916
GALLBLADDER DISEASE, 6420
GASTRITIS
ANTIBIOTICS, 5743
HEPATITIS, CHRONIC, 6299
CHELATING AGENTS, 6293*, 6294*
CORTICOSTEROIDS, 6289*, 6294*, 6338*
IMMUNOSUPPRESSION, 6289*, 6294*
REVIEWS, 6302
VITAMIN B6, 6294*
HEPATITIS, VIRAL
CHILDREN, 6259
HEPATORENAL SYNDROME, 6086*
IRRITABLE COLON, 5916
LIVER
ABSCESS, 6551*
LIVER COMA, 5652
LIVER DISEASE, 6207
PREVENTION, 6175
VITAMIN B12, 6136
LIVER INJURY
ALCOHOL, 5548
CARBON TETRACHLORIDE, 5548
MALABSORPTION, 5916
PANCREATITIS
ANTIENZYMES, 6065, 6073
PHOSPHOLIPASE, 6046*
PARASITES AND PARASITIC DISEASE, 6545*, 6561, 6562
PEPTIC ULCER, 5805, 5818, 5834, 5858
ANTICHOLINERGIC AGENTS, 5821
CARBENOXALONE, 5799*
COMPLICATIONS, 5799*
OXYGEN, 5832
TERPENES, 5843
SALMONELLOSIS
ANTIBACTERIALS, 6540
ANTIBIOTICS, 6540
STOMACH
ULCER, 5319
ULCERATIVE COLITIS, 6020
ANTIBACTERIALS, 6019, 6023

DUMPING SYNDROME
BLOOD
GLUCOSE, 5739*
KININS, 5758
SEROTONIN, 5739*
SURGERY, 5772

DUODENITIS
CHILDREN
PATHOLOGY, 5874
GASTRIN
STOMACH, 5748

DUODENUM
SEE ALSO SMALL INTESTINE
ACIDITY
CHOLECYSTOKININ, 5574*
MOTILIN, 5273*
ARTERY
ANEURYSM, 6542
ANOMALY, 5606
BILIARY DISEASE
COMPLICATIONS, 5891
BLEEDING
DIAGNOSIS, 5660
DRUG-INDUCED, 5660
EPIDEMIOLOGY, 5660
SURGERY, 6500
TRACER STUDY, 5633*
CALCIUM
PANCREATITIS, CHRONIC, 6051*
PARATHYROID GLAND, 6051*
DISEASES
ENDOSCOPY, 5667
RADIOLOGY, 5667
DIVERTICULUM, 5872
RADIOLOGY, 5863*
SURGERY, 5863*
ULCER, 5836
DYSKINESIA
SEDATIVES, 5882
FATTY ACIDS
FEEDING, 5286*
GASTRIN, 5286*
FOREIGN BODIES
DIAGNOSIS, 5765
THERAPY, 5765
GASTRIN
AGE FACTORS, 5206*
EMBRYOLOGY, 5206*
HEMATOMA
COMPLICATIONS, 5880
LACTOFERRIN
PANCREATITIS, CHRONIC, 6050*
MAGNESIUM
PANCREATITIS, CHRONIC, 6051*
PARATHYROID GLAND, 6051*
MOTILITY
GLUCAGON, 5626*
NEOPLASMS, MALIGNANT
CHEMOTHERAPY, 6484*
DIAGNOSIS, 6484*
ENDOSCOPY, 5897
OBSTRUCTION
BILIARY DISEASE, 5891
PATHOLOGY
CELIAC DISEASE, 5909*
PANCREATITIS, CHRONIC, 6069
PRESSURE STUDIES
CHOLECYSTITIS, 6447
RUPTURE
TRAUMA, 5890
STENOSIS
REGIONAL ENTERITIS, 6592
STOMACH
MOTILITY, 5275*

DUODENUM (continued)
TRIGLYCERIDE
FEEDING, 5286*
GASTRIN, 5286*
TRYPSIN
GIARDIASIS, 6544*
ULCER
DIAGNOSIS, 5827
ENDOSCOPY, 5826
ULTRASTRUCTURE
PROTEIN, 5565*
TECHNIQUES, 5231

DYES
EXCRETION
LIVER, 5547
SMALL INTESTINE
DRUG ABSORPTION, 5262

DYSENTERY
VACCINE
IMMUNITY, 6530, 6532

DYSKINESIA
BILIARY
REVIEWS, 6426
DUODENUM
SEDATIVES, 5882
GASTROINTESTINAL
REVIEWS, 6514

DYSPEPSIA
DRUG TREATMENT, 5916

ECHINOCOCCOSIS
BLOOD
IMMUNOGLOBULINS, 6550*
CYSTS
LIVER, 6570
RUPTURE, 6570
LIVER
ANGIOGRAPHY, 5677
SURGERY, 6556

ECHOGRAPHY
BILE DUCT
CYSTS, 6398*
CIRCULATION
INTESTINE, 5585
LIVER
CYSTS, 5636*
LIVER INJURY
OCCUPATIONAL FACTORS, 6224*
VINYL CHLORIDE, 6224*
PANCREAS, 5702

ELECTROLYTE
COLON
MOTILITY, 5268*
ILEUM
MOTILITY, 5268*

EMBRYOLOGY
ANTRUM
GASTRIN, 5206*
DUODENUM
GASTRIN, 5206*
PANCREAS
GASTRIN, 5206*

EMESIS
SEE VOMITING

EMPHYSEMA
CHOLECYSTITIS, 6382*

ENDOCRINE SYSTEM
MALABSORPTION
ETIOLOGY, 5926
RECTUM
ULTRASTRUCTURE, 5228
SMALL INTESTINE
MORPHOLOGY, 5198*
REVIEWS, 5220
STOMACH
AGE FACTORS, 5334
GASTROINTESTINAL DISEASE, 5216*
MORPHOLOGY, 5225
ULTRASTRUCTURE, 5216*

ENDOMETRIOSIS
SMALL INTESTINE
OBSTRUCTION, 5862*

ENDOSCOPY
ANTRUM, 5695
COLITIS, GRANULOMATOUS, 5622*
COLON
COMPLICATIONS, 6006
NEOPLASMS, MALIGNANT, 5692
POLYPS, 5980
REVIEWS, 5698
DUODENUM
DISEASES, 5667
NEOPLASMS, MALIGNANT, 5697
ULCER, 5826
ESOPHAGUS
ANOMALY, 5728
HYPERPLASIA, 5704*
SCLERODERMA, 5650
TECHNIQUES, 5684
GASTROENTEROSTOMY
TECHNIQUES, 5618*
GASTROINTESTINAL
NEOPLASMS, MALIGNANT, 5641
SURGERY, 5654
GASTROINTESTINAL DISEASE, 5697
ILEUM
OBSTRUCTION, 5889
LARGE INTESTINE
DISEASES, 5671
TECHNIQUES, 5684, 5692
PANCREAS
GLUCAGON, 5626*
PAPILLA OF VATER, 5676
PEPTIC ULCER
COMPLICATIONS, 5828
RECTUM
NEOPLASMS, MALIGNANT, 5692
SMALL INTESTINE
TECHNIQUES, 5684
STOMACH
CHEMICAL BURNS, 5754
EROSIONS, 5785
NEOPLASMS, BENIGN, 5615*
NEOPLASMS, MALIGNANT, 5619*, 5641, 5651, 5740, 5763
TECHNIQUES, 5684
ULCER, 5764
ULCERATIVE COLITIS, 5622*

ENDOTOXIN
CYCLIC ADENOSINE MONOPHOSPHATE
LIVER, 5507
LIVER
NECROSIS, 5397*
LIVER INJURY
PORTACAVAL SHUNT, 5397*
PORTAL HYPERTENSION, 5397*
STOMACH
MORPHOLOGY, 5207*
PERMEABILITY, 5207*

ENTERECTOMY
BILE
SECRETION, 5425
PANCREAS
SECRETION, 5425
SMALL INTESTINE
HYPERTROPHY, 5571*

ENTERITIS
SEE ALSO REGIONAL ENTERITIS
ESCHERICHIA COLI
VACCINE, 6528
ETIOLOGY
ENVIRONMENTAL FACTORS, 6504
REVIEWS, 6504
VACCINE, 6504
INFECTION, VIRUS
MALABSORPTION, 5904*
PROTEIN-LOSING ENTEROPATHY, 5904*
STARCH
HYDROLYSIS, 5865*

ENTEROCOLITIS
DRUG-INDUCED
ANTIBIOTICS, 6534

ENTEROKINASE
SMALL INTESTINE
MORPHOLOGY, 5214*

EOSINOPHILS
GASTROENTERITIS
IMMUNOGLOBULINS, 6480*

EPIDEMIOLOGY
COLON
DISEASES, 5984
DUODENUM
BLEEDING, 5660
ESOPHAGUS
NEOPLASMS, MALIGNANT, 5703*, 5727
HEPATITIS, VIRAL, 6272, 6275, 6280
ANTIGEN, AUSTRALIA, 6253
INFLAMMATORY BOWEL DISEASE
REVIEWS, 6520
LARGE INTESTINE
POLYPS, 5990
LIVER
NEOPLASMS, MALIGNANT, 6143
LIVER DISEASE, ALCOHOLIC, 6272, 6339*
PARASITES AND PARASITIC DISEASE
CHILDREN, 6572
INTESTINE, 6567, 6573
PEPTIC ULCER
ADOLESCENTS, 5811
CHILDREN, 5811

EPIDEMIOLOGY (continued)
RECTUM
DISEASES, 5984
REYES SYNDROME, 6093*
STOMACH
BLEEDING, 5660

EROSIONS
STOMACH
ADRENERGIC BLOCKING AGENTS, 5798
ASPIRIN, 5798
BILE ACIDS, 5255
BLEEDING, 5794
ENDOSCOPY, 5785
HYPERPLASIA, 5785
PREVENTION, 5255
STRESS, 5318

ERYTHROCYTE
LIPIDS
CHOLESTASIS, 6114*
CIRRHOSIS, 6114*
LIVER DISEASE, 6114*

ESCHERICHIA COLI
DIARRHEA
SEROLOGY, 6486*
ENTERITIS
VACCINE, 6528

ESOPHAGITIS, REFLUX
ETIOLOGY, 5706
HERNIA, HIATUS
DISEASES ASSOCIATED WITH, 5729
SURGERY, 5706

ESOPHAGUS
ANOMALY
ENDOSCOPY, 5728
ATRESIA
DISEASES ASSOCIATED WITH, 5724
FAMILIAL FACTORS, 5724
RADIOLOGY, 5722, 5733
SURGERY, 5719, 5722
BIOPOTENTIALS
NERVOUS SYSTEM, 5280
COLITIS
ISCHEMIA, 5985
DISEASES, 5713
ENDOSCOPY
TECHNIQUES, 5684
GASTRECTOMY
SEQUELAE, 5723
HETEROTOPIA
GASTRECTOMY, 5723
HYPERPLASIA
ENDOSCOPY, 5704*
GLYCOGEN, 5704*
MOTILITY
ANTIDIARRHEALS, 5277
TRYPANOSOMIASIS, 6547*
NEOPLASMS, MALIGNANT
DIAGNOSIS, 5709
DISEASES ASSOCIATED WITH, 5705*, 5717, 5732
EPIDEMIOLOGY, 5703*, 5727
HETEROTOPIA, 5705*
PATHOLOGY, 5710
RADIOTHERAPY, 5710, 5715
SURGERY, 5711, 5720, 5731

ESOPHAGUS (continued)
PERFORATION
DISEASES ASSOCIATED WITH, 6522
RADIOLOGY
PYLOROPLASTY, 5836
VAGOTOMY, 5836
VOMITING, 5716
RUPTURE
THERAPY, 5726
SCLERODERMA
ENDOSCOPY, 5650
RADIOLOGY, 5650
SPHINCTER
PRESSURE STUDIES, 5306*, 5713
STENOSIS
DISEASES ASSOCIATED WITH, 5730
REFLUX, 5714
SURGERY, 5714, 5720, 5730
SURGERY
COLITIS, 5985
COMPLICATIONS, 5985
REFLUX, 5718
VARICES
CIRRHOSIS, 6336*
REVIEWS, 6336*
THERAPY, 6336*

ESTROGENS
BILE
EXCRETION, 5459
BLOOD
LIVER DISEASE, ALCOHOLIC, 6317*
METABOLISM
LIVER, 5459
URINE
LIVER DISEASE, ALCOHOLIC, 6317*

ETHNIC FACTORS
LACTOSE TOLERANCE, 5924
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 5948
LIVER
NEOPLASMS, MALIGNANT, 6143

EXCRETION
ANTIBIOTICS
BILE, 6401*
BILE
ANTICOAGULANTS, 5453
BILE ACIDS, 6102*
CARBON TETRACHLORIDE, 5500
ESTROGENS, 5459
SULFOBROMOPHTHALEIN, 5410*, 5411*
CHOLESTEROL
BILE, 5424
BILE ACIDS, 5424
CONTRAST MEDIA
CHOLERETIC AGENTS, 5362*
LIVER, 5362*, 5683
DYES
LIVER, 5547
PHOSPHOLIPID
BILE, 5424
BILE ACIDS, 5424
WATER
CIRRHOSIS, 6340*

FAMILIAL FACTORS
CHOLELITHIASIS
CHILDREN, 6439

FAMILIAL FACTORS (continued)
CIRRHOSIS
ETIOLOGY, 6318*
CROHNS DISEASE, 6584
ESOPHAGUS
ATRESIA, 5724
LARGE INTESTINE
POLYPS, 5928*, 5931*
PANCREATITIS
AMINO ACIDS, 6045*

FASCIOLIASIS
BILE DUCT
DRUG TREATMENT, 6557

FATS
SEE LIPIDS, TRIGLYCERIDES, GLYCOLIPIDS, PHOSPHOLIPID

FATTY ACIDS
BLOOD
TRIGLYCERIDE, 5435
DEFICIENCY
STOMACH, 5281
DUODENUM
FEEDING, 5286*
GASTRIN, 5286*
JEJUNUM
ABSORPTION, 5246*
LIVER
DIETARY FACTORS, 5433
GLUCONEOGENESIS, 5363*
TRIGLYCERIDE, 5435
METABOLISM
LIVER, 5363*
OXIDATION
LIVER, 5551
PERITONEUM
PATHOLOGY, 5209*
SMALL INTESTINE
PATHOLOGY, 5209*
SYNTHESIS
CYCLIC ADENOSINE MONOPHOSPHATE, 5540
LIVER, 5546

FATTY LIVER
ALCOHOL
TRIGLYCERIDE, 5376*
ALCOHOLISM
REVIEWS, 6327
CHOLESTEROL, 5536
CORTICOSTEROIDS
METABOLISM, 6127*
DIETARY FACTORS
CHOLESTEROL, 5473
DRUG-INDUCED
AMINO ACIDS, 5474
ANTIBIOTICS, 5559
ETIOLOGY
PREVENTION, 5560
GLUCOSE
METABOLISM, 5474
HORMONE EFFECTS ON, 5560
LIVER
REGENERATION, 5422
OBESITY
SHUNT, INTESTINAL, 6111*, 6116*

FATTY LIVER (continued)
SHUNT, INTESTINAL
OBESITY, 5570*
THERAPY
REVIEWS, 6163

FECES
CHEMICAL COMPOSITION
GASTROENTEROSTOMY, 5814
PEPTIC ULCER, 5814
SALMONELLOSIS
IMMUNOLOGY, 6518

FEEDING
ALCOHOL
METABOLISM, 6315*
BILIRUBIN
BINDING, 6215*
BLOOD
GASTRIN, 5313
CHYME
NITROGEN, 5290
DUODENUM
FATTY ACIDS, 5286*
TRIGLYCERIDE, 5286*
GASTRIN
BLOOD, 5839
SMALL INTESTINE
BIOPOTENTIALS, 5259
STOMACH
ACID SECRETION, 5313

FETOPROTEIN, ALPHA
BLOOD
LIVER DISEASE, 6117*
HEPATITIS, CHRONIC, 6309
LIVER
NEOPLASMS, MALIGNANT, 5417*, 6131, 6195
REGENERATION, 5534, 6117*
LIVER DISEASE, 6094*
CHILDREN, 6189
PANCREAS
NEOPLASMS, MALIGNANT, 6033

FIBER
LIPIDS
METABOLISM, 5538

FIBRINOGEN
METABOLISM
HEPATITIS, CHRONIC, 6238*
HEPATITIS, VIRAL, 6238*
SYNTHESIS
PORTAL HYPERTENSION, 6343*

FIBRINOLYSIS
BLOOD
BILIARY DISEASE, 6188
LIVER DISEASE, 6188

FIBRINOLYTIC AGENTS
LIVER FUNCTION TESTS
DRUG EFFECTS ON, 6179

FIBROSIS
BILE DUCT
LIVER, 6428

FIBROSIS (continued)
LIVER
ANOMALY, CONGENITAL, 6121*
COLLAGEN, 6119*
CONNECTIVE TISSUE, 6119*
SCHISTOSOMIASIS, 6003

FISTULA
BILIARY
SURGERY, 6395*, 6459
CHOLECYSTITIS
COMPLICATIONS, 6438
COLON
STOMACH DISEASE, 5790
GALLBLADDER
DIAGNOSIS, 6442
STOMACH, 6442
THERAPY, 6442
RECTUM
NEOPLASMS, MALIGNANT, 5950
SMALL INTESTINE
CROHNS DISEASE, 6593
SURGERY, 6395*
STOMACH
COLON, 5768
ULCER, 5790

FLUID
GASTROINTESTINAL
IMMUNOGLOBULINS, 5596*

FOLIC ACID
METABOLISM
ANTICONVULSANTS, 5482
LIVER, 5454, 5482

FOOD
SALMONELLOSIS
IMMUNOLOGY, 6518

FOOD ADDITIVES
LIVER
PHOSPHORYLATION, OXIDATIVE, 5450

FOREIGN BODIES
DUODENUM
DIAGNOSIS, 5765
THERAPY, 5765
GASTROINTESTINAL
CHILDREN, 6476*
RADIOLOGY, 6476*
STOMACH
DIAGNOSIS, 5765
NEOPLASMS, MALIGNANT, 5783
PEPTIC ULCER, 5813
THERAPY, 5765

FRUCTOSE
GLYCOGEN STORAGE DISEASE
LIVER, 5218
LIVER
PATHOLOGY, 5218

GALACTOSAMINE
LIVER
PHOSPHOLIPID, 5503
ULTRASTRUCTURE, 5503

GALACTOSAMINE (continued)
LIVER INJURY
ALBUMIN, 5446
CLOTTING, 5446
DRUG-INDUCED, 5385*

GALLBLADDER
ANOMALY, CONGENITAL, 6431
DIVERTICULUM, 6430
FISTULA
DIAGNOSIS, 6442
STOMACH, 6442
THERAPY, 6442
MOTILITY
ANTIEMETICS, 6467
GASTRECTOMY, 5749
RADIOLOGY, 6467
VAGOTOMY, 5749
NEOPLASMS, BENIGN
DISEASES ASSOCIATED WITH, 6464
NEOPLASMS, MALIGNANT, 6423
PATHOLOGY, 6399*
SURGERY, 6399*, 6407
SURVIVAL, 6407
PATHOLOGY
CHOLELITHIASIS, 5230
MORPHOLOGY, 5230
PERFORATION
ANGIOGRAPHY, 6452
COMPLICATIONS, 6452
SURGERY, 6475
SURGERY
COMPLICATIONS, 6458
ULTRASTRUCTURE
CHOLECYSTITIS, 6394*
CHOLELITHIASIS, 6394*
MUCUS, 5236

GALLBLADDER DISEASE, 6173
ANGIOGRAPHY
TECHNIQUES, 6472
CHOLECYSTECTOMY, 6389*
DRUG TREATMENT, 6420
RADIOLOGY, 5662, 5674, 5678, 6389*
SURGERY, 6441

GALLSTONE
SEE ALSO CHOLELITHIASIS,
CHOLEDOCHOLITHIASIS
ILEUS, 5873
SURGERY, 5864*
MORPHOLOGY, 6440

GASTRECTOMY
SEE ALSO ANTRECTOMY
BLOOD
CALCIUM, 5320
COLON
ANOMALY, CONGENITAL, 5954
HERNIA, 5954
ESOPHAGUS
HETEROTOPIA, 5723
GALLBLADDER
MOTILITY, 5749
GASTROINTESTINAL
BLEEDING, 5810
GLUCOSE
ABSORPTION, 5774

GASTRECTOMY (continued)
LIPIDS
ABSORPTION, 5774
PANCREAS
SECRETION, 5806
SEQUELAE
ESOPHAGUS, 5723
STOMACH
ACIDITY, 5825
NEOPLASMS, MALIGNANT, 5745
VITAMIN B12
ABSORPTION, 5759

GASTRIN
ANTRUM
AGE FACTORS, 5206*
EMBRYOLOGY, 5206*
BLOOD
ACIDITY, 5306*, 5310*
ALCOHOLISM, 5304*
ANTACIDS, 5838
CALCIUM, 5307*, 5310*, 5320
FEEDING, 5313, 5839
MAGNESIUM, 5307*
PEPTIC ULCER, 5804, 5838, 5839
RADIOIMMUNOASSAY, 5685
VAGOTOMY, 5804
ZOLLINGER-ELLISON SYNDROME, 6483*
6521
DUODENUM
AGE FACTORS, 5206*
EMBRYOLOGY, 5206*
FATTY ACIDS, 5286*
TRIGLYCERIDE, 5286*
GASTROINTESTINAL
MORPHOLOGY, 5285*
PANCREAS
AGE FACTORS, 5206*
EMBRYOLOGY, 5206*
NEOPLASMS, 5593*, 6483*
SECRETION
ALCOHOL, 5348
SMALL INTESTINE
ENZYMES, 5583
STOMACH
ACID SECRETION, 5321, 5322
CHOLECYSTOKININ, 5321
DUODENITIS, 5748
GASTRITIS, 5748
HYPERCHLORHYDRIA, 5748
IMMUNOLOGY, 5331
MORPHOLOGY, 5225
PEPTIC ULCER, 5748
SECRETION, 5331, 5335

GASTRITIS
AUTOIMMUNITY, 5761
DISEASES ASSOCIATED WITH
IMMUNOLOGY, 5761
DRUG TREATMENT
ANTIBIOTICS, 5743
GASTRIN
STOMACH, 5748
PATHOLOGY
NERVOUS SYSTEM, 5747
STOMACH
SECRETION, 6489

GASTRITIS, ATROPHIC
ETIOLOGY
IMMUNOLOGY, 5760
PANCREAS
SECRETION, 6029*
SECRETION
ANTICHOLINERGIC AGENTS, 5741
STEROID, 5741
VITAMINS, 5741

GASTRODUODENITIS
CHILDREN
DIAGNOSIS, 6488
IMMUNOGLOBULINS, 6488

GASTROENTERITIS
ALLERGY
DIETARY FACTORS, 6480*
EOSINOPHILS
IMMUNOGLOBULINS, 6480*
INTESTINE
MOTILITY, 6477*
JEJUNUM
VIRUS, 6536
THERAPY
DIET, 6507
NEONATE, 6507

GASTROENTEROSTOMY
ENDOSCOPY
TECHNIQUES, 5618*
FECES
CHEMICAL COMPOSITION, 5614
HEMATOPOIESIS, 6500
PANCREAS
CIRCULATION, 6030*
PANCREAS FUNCTION TESTS, 6048*
SEQUELAE
PANCREAS DISEASE, 6030*

GASTROINTESTINAL
ACIDITY
ILEUS, 5877
ALBUMIN
METABOLISM, 5293
BIOPOTENTIALS
PEPTIDES, 5270*
BLEEDING
ANTACIDS, 6479*
ANTIINFLAMMATORY AGENTS, 6492
ASPIRIN, 6154, 6479*
CIRRHOSIS, 6365, 6375
DRUG-INDUCED, 6479*, 6492
ETIOLOGY, 6501
GASTRECTOMY, 5810
LIVER FUNCTION TESTS, 6365
REVIEWS, 6508
THERAPY, 6501
CARCINOIDS
DISEASES ASSOCIATED WITH, 6487
NEOPLASMS, MALIGNANT, 6487
DYSKINESIA
REVIEWS, 6514
FLUID
IMMUNOGLOBULINS, 5596*
FOREIGN BODIES
CHILDREN, 6476*
RADIOLOGY, 6476*
IMMUNOGLOBULINS, 5596*

GASTROINTESTINAL (continued)
INFLAMMATION
ETIOLOGY, 5600*
MORPHOLOGY
GASTRIN, 5285*
HYPERALIMENTATION, 5285*
MOTILITY
ALCOHOL, 5254
CHOLERETIC AGENTS, 5289
DRUG EFFECTS ON, 5254, 5652
HORMONE CONTROL, 5279
NERVOUS CONTROL, 5278
REVIEWS, 5279
NEOPLASMS, MALIGNANT
ANTIGEN, CARCINOEMBRYONIC, 5688
COMPLICATIONS, 6516
ENDOSCOPY, 5641
IMMUNOLOGY, 6498
IRRADIATION, 5613
NITROSAMINES, 5613
PERFORATION, 6516
RADIOTHERAPY, 6499
NUCLEOTIDASE, 5292
PARASITES AND PARASITIC DISEASE
COMPLICATIONS, 6559
PROTEINASE
VITAMIN B6, 5610
SCLERODERMA
SURGERY, 6541
SECRETION
CHOLERETIC AGENTS, 5289
SURGERY
ENDOSCOPY, 5654
KIDNEY DISEASE, 6503
PANCREATITIS, 6077
TUBERCULOSIS
COMPLICATIONS, 5986
RADIOLOGY, 5986

GASTROINTESTINAL DISEASE
ENDOSCOPY, 5697
IMMUNOLOGY
REVIEWS, 6506
SCANNING, SCINTILLATION, 5660
STOMACH
ENDOCRINE SYSTEM, 5216*
TRACER STUDY, 5680

GASTROINTESTINAL TRACT
ALCOHOL
ABSORPTION, 5254
BLEEDING
TRACER STUDY, 5633*
HORMONE
HYPERALIMENTATION, 5591*
LIVER
METASTASES, 6171
MITOSIS
HYPERALIMENTATION, 5595*
MORPHOLOGY
HYPERALIMENTATION, 5591*, 5595*
NEOPLASMS, MALIGNANT
METASTASES, 6171
VITAMIN B6
DEFICIENCY, 5610

GENETIC FACTORS
ALCOHOLISM
ETIOLOGY, 5361*

GENETIC FACTORS (continued)
BLOOD
TRANSAMINASE, 5441
LIVER
PATHOLOGY, 6098*

GENETICS
CIRRHOSIS
CHILDREN, 6333*
PANCREAS
AMYLASE, 5344*
ISOENZYMES, 5344*
TRANSPLANTATION, 5346

GEOGRAPHICAL FACTORS
LIVER
NEOPLASMS, MALIGNANT, 6248*

GIARDIASIS
DUODENUM
TRYPSIN, 6544*
PANCREAS
TRYPSIN, 6544*

GILBERTS DISEASE
BILIRUBIN
BINDING, 6215*

GLUCAGON
DUODENUM
MOTILITY, 5626*
PANCREAS
ENDOSCOPY, 5626*
PANCREATITIS
PREVENTION, 5626*

GLUCONEOGENESIS
LIVER
ADRENERGIC BLOCKING AGENTS, 5484
FATTY ACIDS, 5363*
HORMONE CONTROL, 5470
SYMPATHOMIMETIC AGENTS, 5484

GLUCOSAMINE
METABOLISM
ASPIRIN, 5505
LIVER, 5505

GLUCOSE
ABSORPTION
GASTRECTOMY, 5774
VAGOTOMY, 5774
BLOOD
DUMPING SYNDROME, 5739*
INTOLERANCE
HEPATECTOMY, 6096*
JAUNDICE, OBSTRUCTIVE, 5389*
JEJUNUM
OBSTRUCTION, 5580
LIVER
CIRCULATION, 5399*
SURGERY, 5399*
METABOLISM
CIRRHOSIS, 6335*
FATTY LIVER, 5474
KWASHIORKOR, 6543
MARASMUS, 6543
POTASSIUM
TRANSPORT, 5257

GLUCOSE (continued)
SYNTHESIS
LIVER, 5452
PROSTAGLANDIN, 5452
TRANSPORT
GLYCOSIDES, 5250*
LACTASE DEFICIENCY, 5250*
SMALL INTESTINE, 5250*

GLUCOSE-6-PHOSPHATE
DEFICIENCY
DISEASES ASSOCIATED WITH, 6103*
HYPERLIPEMIA, 6103*

GLUCURONIDASE, BETA
LIVER
CRYOSURGERY, 5512

GLUTAMYL TRANSPEPTIDASE, GAMMA
BILE
CHOLESTASIS, 6387*
PANCREAS
CALCULI, 6027*

GLUTATHIONE
BINDING
SULFOBROMOPHTHALEIN, 5410*
CIRRHOSIS
DRUG TREATMENT, 6352
LIVER
ENZYMES, 5476
MITOCHONDRIA, 5497

GLYCOGEN
ESOPHAGUS
HYPERPLASIA, 5704*
LIVER
HEPATECTOMY, 5532

GLYCOGEN STORAGE DISEASE
COLON
RADIOLOGY, 6204
DISEASES ASSOCIATED WITH
COLITIS, 6204
LIVER
FRUCTOSE, 5218

GLYCOPROTEINS
BILE
CHEMICAL COMPOSITION, 5481
LIVER
ISCHEMIA, 6133
ULTRASTRUCTURE, 5449
SYNTHESIS
SMALL INTESTINE, 5563*

GLYCOSIDES
ABSORPTION
MALNUTRITION, 6485*
MICROORGANISMS, 6485*
BILIRUBIN
BILE, 6213*
CHROMATOGRAPHY, 6213*
GLUCOSE
TRANSPORT, 5250*

GNOTOBIOSIS
IMMUNOGLOBULINS
SMALL INTESTINE, 5604

GROWTH FACTOR
ILEUM
SHUNT, INTESTINAL, 5224

GROWTH RETARDATION
VITAMIN B12
MALABSORPTION, 5918

H2 RECEPTOR ANTAGONISTS
SMALL INTESTINE
MOTILITY, 5564*
STOMACH
ACID SECRETION, 5309*, 5324
SECRETION, 5332

HAMARTOMA
BILIARY
SURGERY, 6448

HEARTBURN
DISEASES ASSOCIATED WITH
PEPITC ULCER, 5831

HEIDENHAIN POUCH
STOMACH
SECRETION, 5305*

HELMINTHIASIS
DRUG EFFECTS ON
ANTIBIOTICS, 6576

HEMANGIOMA
LIVER
COMPLICATIONS, 6172
DIAGNOSIS, 6081*
NEONATE, 6081*
SURGERY, 6136, 6172

HEMATOMA
DUODENUM
COMPLICATIONS, 5880
LIVER
DRAINAGE, 6203

HEMATOPOIESIS
GASTROENTEROSTOMY, 6500

HEMATOPOIETIC SYSTEM
ULCERATIVE COLITIS
COMPLICATIONS, 6021

HEME
METABOLISM
COBALT, 5356*
LIVER, 5356*

HEMOBILIA
DIAGNOSIS, 6436
THERAPY, 6436

HEMOCHROMATOSIS
ALCOHOLISM
REVIEWS, 6329
PANCREAS
SECRETION, 6041
THERAPY
REVIEWS, 6164

HEMORRHAGE
SEE BLEEDING

HEPATECTOMY
BILE DUCT
DILATATION, 6453
COMPLICATIONS
PREVENTION, 6205
GLUCOSE
INTOLERANCE, 6096*
LIVER
ABSCESS, 6152
CYSTS, 6152
ENZYMES, 5418*
GLYCOGEN, 5532
LIPIDS, 5532
MITOSIS, 5532
MORPHOLOGY, 5508
NEOPLASMS, MALIGNANT, 6152
TRAUMA, 6152
ULTRASTRUCTURE, 5418*
NUCLEIC ACIDS
SYNTHESIS, 5357*, 5392*, 5556
STOMACH
ACID SECRETION, 5297*
TECHNIQUES
IRRADIATION, 5510

HEPATITIS
BLOOD
LIPOPROTEINS, 5624*
DRUG-INDUCED
AUTOIMMUNITY, 6101*
NEONATE
LIPOPROTEINS, 5624*
PATHOLOGY, 6211*
PROGNOSIS, 6211*
VIRUS
LIVER FUNCTION TESTS, 5499
PATHOLOGY, 5499

HEPATITIS, ACUTE
PROGNOSIS
IMMUNOGLOBULINS, 6243*

HEPATITIS, CHRONIC
ANTIGEN, AUSTRALIA, 6245*
CORTICOSTEROIDS, 6306
IMMUNOLOGY, 6273
KIDNEY DISEASE, 6295*
METABOLISM, 6288
AUTOIMMUNITY
REVIEWS, 6101*
BLOOD
PHYSICAL PROPERTIES, 6338*
CHILDREN
IMMUNOSUPPRESSION, 6291*
STEROID, 6291*
THERAPY, 6296
COMPLEMENT
ANTIGEN, AUSTRALIA, 6290*
COMPLICATIONS
CIRRHOSIS, 6297
CORTICOSTEROIDS
METABOLISM, 6097*, 6127*
DIAGNOSIS, 6298
IMMUNOGLOBULINS, 6294*
REVIEWS, 6307

HEPATITIS, CHRONIC (continued)
DRUG TREATMENT, 6299
CHELATING AGENTS, 6293*, 6294*
CORTICOSTEROIDS, 6289*, 6294*, 6338*
IMMUNOSUPPRESSION, 6289*, 6294*
REVIEWS, 6302
VITAMIN B6, 6294*
ETIOLOGY
IMMUNITY, 6298
FETOPROTEIN, ALPHA, 6309
FIBRINOGEN
METABOLISM, 6238*
IMMUNOGLOBULINS, 6243*, 6245*
IMMUNOLOGY, 6292*
LIPIDS
METABOLISM, 6301
LIVER
DRUG METABOLISM, 6310
NEOPLASMS, MALIGNANT, 6304
NUCLEIC ACIDS
SYNTHESIS, 6244*, 6292*
PROGNOSIS, 6310
REVIEWS, 6303, 6305
THERAPY, 6308, 6523
REVIEWS, 6187, 6300, 6307
ULCERATIVE COLITIS
DISEASES ASSOCIATED WITH, 6523

HEPATITIS, NONVIRAL
ALCOHOLISM
IMMUNOLOGY, 6328
DIAGNOSIS, 6230
DRUG-INDUCED
ANESTHETICS, 6226, 6230
ANTIBACTERIALS, 6217*
HYDROCARBONS, HALOGENATED, 6226
REVIEWS, 6327

HEPATITIS, SERUM
ANTIGEN, AUSTRALIA
CHILDREN, 6260

HEPATITIS, VIRAL
AMINO ACIDS
METABOLISM, 6283
ANTIGEN, AUSTRALIA, 6245*
AGE FACTORS, 6250
CARRIER STATE, 5466
CHILDREN, 6260
DIAGNOSIS, 6278
EPIDEMIOLOGY, 6253
IMMUNITY, 6237*
IMMUNOLOGY, 5466, 6273, 6279
LIVER FUNCTION TESTS, 6274
REVIEWS, 6277
SEROLOGICAL DIAGNOSIS, 6276
AUTOIMMUNITY
REVIEWS, 6101*
BIOPSY, 6263
BLOOD
PERFUSION, 6239*
CHILDREN, 6252
DRUG TREATMENT, 6259
HYDROCARBONS, 6259
SEQUELAE, 6270
CIRRHOSIS
CHILDREN, 6246*

HEPATITIS, VIRAL (continued)
COMPLICATIONS
KIDNEY DISEASE, 6264
PREVENTION, 6262
REVIEWS, 6255
DIAGNOSIS, 6269, 6282
ALDOLASE, 6268
CHILDREN, 6268
ETIOLOGY, 6261
DISEASES ASSOCIATED WITH, 6258
CIRCULATION, 6287
RESPIRATORY SYSTEM, 6287
EPIDEMIOLOGY, 6272, 6275, 6280
ETIOLOGY, 6285
FIBRINOGEN
METABOLISM, 6238*
HYPOGLYCEMIA, 6267
IMMUNITY, 6249*
IMMUNOGLOBULINS, 6245*
IMMUNOLOGY
CHILDREN, 6284
LAPAROSCOPY, 6263
LEUKOCYTES
PHAGOCYTOSIS, 6254
LIVER
ANTIGEN, AUSTRALIA, 6251, 6286
CHOLESTASIS, 6240*
CHOLESTEROL, 6240*
NEOPLASMS, MALIGNANT, 6248*
PATHOLOGY, 6240*
TISSUE CULTURE, 6279
LIVER FUNCTION TESTS
AGE FACTORS, 6281
NUCLEIC ACIDS
SYNTHESIS, 6244*
PREVENTION, 6256, 6280
TRANSFUSION, 6242*
PROGNOSIS, 6285
RECURRENCE
SEROLOGICAL DIAGNOSIS, 6235*
RISK FACTORS
ANTIGEN, AUSTRALIA, 6242*
SEQUELAE
CIRRHOSIS, 6265
REVIEWS, 6265
THERAPY, 6269
REVIEWS, 6257
TRACE ELEMENTS
BALANCE STUDIES, 6234*
CHILDREN, 6234*
TRANSFUSION
ETIOLOGY, 6266
UREA
METABOLISM, 6283
VACCINE
COMPLICATIONS, 6241*

HEPATORENAL SYNDROME
DRUG EFFECTS ON, 6346*
DRUG TREATMENT, 6086*
KIDNEY
CIRCULATION, 6346*
PORTACAVAL SHUNT, 6086*

HERNIA
COLON
GASTRECTOMY, 5954
LIVER
PROLAPSE, 6166

HERNIA (continued)
SMALL INTESTINE
SURGERY, 5876

HERNIA, HIATUS
DISEASES ASSOCIATED WITH
ESOPHAGITIS, REFLUX, 5729
STRANGULATION
SURGERY, 5708
SURGERY
TECHNIQUES, 5725

HETEROTOPIA
ESOPHAGUS
GASTRECTOMY, 5723
NEOPLASMS, MALIGNANT, 5705*
PANCREAS, 6038

HIRSCHSPRUNGS DISEASE
DEFECATION
RADIOLOGY, 5638*
RADIOLOGY
NEONATE, 5623*

HISTAMINE
PLASMA
PROTEIN, 5311*
SMALL INTESTINE
MOTILITY, 5274*, 5564*
STOMACH
ACID SECRETION, 5298*, 5309*, 5324, 5328, 5734*
SECRETION, 5809

HODGKINS DISEASE
BILIRUBIN
METABOLISM, 6156
COLON, 6010

HORMONE
GASTROINTESTINAL TRACT
HYPERALIMENTATION, 5591*
HYPERBILIRUBINEMIA
DRUG-INDUCED, 6212*
JEJUNUM
HYPERCHLORHYDRIA, 5296*
PEPTIC ULCER
PREVENTION, 5333

HORMONE CONTROL
APPETITE
CHOLECYSTOKININ, 5603
GASTROINTESTINAL
MOTILITY, 5279
GLUCONEOGENESIS
LIVER, 5470
LIVER
PROTEIN, 5426
STOMACH
ACID SECRETION, 5296*

HORMONE EFFECTS ON
FATTY LIVER, 5560
PANCREAS
SECRETION, 5333
SMALL INTESTINE
BIOPOTENTIALS, 5259
CYCLIC GUANOSINE MONOPHOSPHATE, 5575*

HORMONE EFFECTS ON (continued)
STOMACH
SECRETION, 5333

HUMORAL FACTORS
LIVER
REGENERATION, 5400*
PANCREAS
NEOPLASMS, MALIGNANT, 6028*

HYALIN
PANCREAS
ALCOHOLISM, 6052*
LIVER DISEASE, ALCOHOLIC, 6052*

HYDROCARBONS
HEPATITIS, VIRAL
CHILDREN, 6259
LIVER
DRUG METABOLISM, 5553

HYDROCARBONS, HALOGENATED
HEPATITIS, NONVIRAL
DRUG-INDUCED, 6226
LIVER
MORPHOLOGY, 5223
LIVER INJURY
PHENOBARBITAL, 5416*
METABOLISM
LIVER, 5477

HYDROCARBONS, POLYCYCLIC
LIVER
DRUG METABOLISM, 5558, 6150

HYDROLYSIS
MUCOPOLYSACCHARIDE
COLON, 5211*
STARCHES
ENTERITIS, 5865*
PANCREATITIS, CHRONIC, 5865*

HYPERALIMENTATION
GASTROINTESTINAL TRACT
MITOSIS, 5595*
MORPHOLOGY, 5595*
GASTROINTESTINAL
MORPHOLOGY, 5285*
GASTROINTESTINAL TRACT
HORMONE, 5591*
MORPHOLOGY, 5591*
LIVER
PATHOLOGY, 6220*
LIVER FUNCTION TESTS, 6220*
LIVER INJURY
SIMULATION, 6220*

HYPERBILIRUBINEMIA
BLOOD
BILE ACIDS, 6118*
DRUG-INDUCED
HORMONE, 6212*

HYPERCHLORHYDRIA
SEE ALSO ACID SECRETION
DISEASES ASSOCIATED WITH
PEPTIC ULCER, 5831
GASTRIN
STOMACH, 5748

HYPERCHLORHYDRIA (continued)
JEJUNUM
HORMONE, 5296*
STOMACH
MOTILITY, 5769

HYPERLIPEMIA
GLUCOSE-6-PHOSPHATE
DEFICIENCY, 6103*
LIVER
LIPIDS, 6202

HYPERLIPOPROTEINEMIA
PANCREATITIS
DISEASES ASSOCIATED WITH, 6057*
ETIOLOGY, 6057*
STIMULATION
ALCOHOL, 5414*

HYPERPLASIA
ESOPHAGUS
ENDOSCOPY, 5704*
GLYCOGEN, 5704*
KIDNEY
SPLEEN, 5511
LIVER
BLEEDING, 6200
ORAL CONTRACEPTIVES, 6200
STOMACH
EROSIONS, 5785

HYPERTENSION
LIVER FUNCTION TESTS
ATHEROSCLEROSIS, 6155
RADIOISOTOPES, 6155

HYPERTROPHY
ILEUM
SHUNT, INTESTINAL, 5224
LIVER
PORTACAVAL SHUNT, 5364*
PYLORUS
PATHOLOGY, 5779
STENOSIS, 5771, 5791
SMALL INTESTINE
ENTERECTOMY, 5571*
NERVOUS SYSTEM, 5571*

HYPOCHLORHYDRIA
SEE ALSO ACID SECRETION

HYPOCHOLESTEREMIC AGENTS
ADENOSINE TRIPHOSPHATASE
LIVER, 5531

HYPOGLYCEMIA
HEPATITIS, VIRAL, 6267

HYPOTHERMIA
STOMACH
BLEEDING, 5794

HYPOXIA
LIVER
PROTEIN, 5437

ICTERUS
SEE JAUNDICE

ILEOSTOMY
DIARRHEA
PREVENTION, 5861*
ILEUM
ABSORPTION, 5867*
BACTERIA, 5867*
MORPHOLOGY, 5867*
ULCERATIVE COLITIS
DIET, 6022
MICROORGANISMS, 6022
VITAMIN B12
ABSORPTION, 5866*

ILEUM
SEE ALSO SMALL INTESTINE
ABSORPTION
ILEOSTOMY, 5867*
INFLAMMATORY BOWEL DISEASE, 6520
PERFUSION, 5249*
TECHNIQUES, 5249*
BACTERIA
ILEOSTOMY, 5867*
GROWTH FACTOR
SHUNT, INTESTINAL, 5224
HYPERTROPHY
SHUNT, INTESTINAL, 5224
ISCHEMIA
SCANNING, SCINTILLATION, 5605
METALS
CADMIUM, 5526
MORPHOLOGY
ILEOSTOMY, 5867*
MOTILITY
CALCIUM, 5268*, 5283
DRUG-INDUCED, 5283
ELECTROLYTE, 5268*
SODIUM, 5283
OBSTRUCTION
ENDOSCOPY, 5889
THERAPY, 5889
PERFORATION
DISEASES ASSOCIATED WITH, 6522
SODIUM
TRANSPORT, 5242*, 5256
SURGERY
DIARRHEA, 5893
SEQUELAE, 5893

ILEUS
GALLSTONE, 5873
SURGERY, 5864*
GASTROINTESTINAL
ACIDITY, 5877
PREVENTION, 5971
RADIOLOGY, 5937*
SURGERY, 5885
THERAPY, 5885

IMMUNITY
ANTIGEN, AUSTRALIA
CARRIER STATE, 6237*
CHOLERA
VACCINE, 6527
DYSENTERY
VACCINE, 6530, 6532
HEPATITIS, CHRONIC
ETIOLOGY, 6298

IMMUNITY (continued)
HEPATITIS, VIRAL, 6249*
ANTIGEN, AUSTRALIA, 6237*
SALMONELLOSIS
VACCINE, 6510, 6511, 6529
SHIGELLOSIS
VACCINE, 6531
SMALL INTESTINE
RADIOLOGY, 5888

IMMUNOGLOBULINS
BLOOD
CHOLERA, 6513
ECHINOCOCCOSIS, 6550*
PANCREAS DISEASE, 6055*
PANCREATITIS, 6055*
SCHISTOSOMIASIS, 6550*
TOXOPLASMOSIS, 6550*
TRICHINOSIS, 6550*
CIRRHOSIS, 6245*
DEFICIENCY
LIVER COMA, 6112*
MALABSORPTION, 5903*, 5914
PROTEIN-LOSING ENTEROPATHY, 5903*
GASTRODUODENITIS
CHILDREN, 6488
GASTROENTERITIS
EOSINOPHILS, 6480*
GASTROINTESTINAL, 5596*
FLUID, 5596*
HEPATITIS, ACUTE
PROGNOSIS, 6243*
HEPATITIS, CHRONIC, 6243*, 6245*
DIAGNOSIS, 6294*
HEPATITIS, VIRAL, 6245*
LIVER DISEASE
AUTOIMMUNITY, 6082*
LIVER DISEASE, ALCOHOLIC
DIAGNOSIS, 6320
PROGNOSIS, 6320
PANCREAS
SECRETION, 6026*
TRAUMA, 6026*
SMALL INTESTINE
AGE FACTORS, 5604
GNOTOBIOSIS, 5604
STOMACH
NEOPLASMS, MALIGNANT, 5795

IMMUNOSUPPRESSION
CIRRHOSIS
DRUG TREATMENT, 6289*
HEPATITIS, CHRONIC
CHILDREN, 6291*
DRUG TREATMENT, 6289*, 6294*
LIVER FUNCTION TESTS
DRUG EFFECTS ON, 6247*

IMMUNOTHERAPY
STOMACH
SECRETION, 5829

INDOCYANINE GREEN
LIVER DISEASE
DIAGNOSIS, 6079*

INFECTION, BACTERIA
BILIARY TRACT
ANTIBIOTICS, 6424
DIAGNOSIS, 6424
INTESTINE
VACCINE, 6533
NUCLEIC ACIDS
SYNTHESIS, 5490
SMALL INTESTINE
SURGERY, 5892

INFECTION, VIRUS
BILIARY
OBSTRUCTION, 5401*
ENTERITIS
MALABSORPTION, 5904*
PROTEIN-LOSING ENTEROPATHY, 5904*
LIVER
PATHOLOGY, 6144
REYES SYNDROME, 6093*, 6197

INFLAMMATION
GASTROINTESTINAL
ETIOLOGY, 5600*

INFLAMMATORY BOWEL DISEASE
EPIDEMIOLOGY
REVIEWS, 6520
ILEUM
ABSORPTION, 6520
IMMUNOLOGY
REVIEWS, 6520
LARGE INTESTINE
THERAPY, 6585

INSECTICIDES
LIVER
DRUG METABOLISM, 5554
METABOLISM
LIVER, 5477, 5530

INTEGUMENTARY SYSTEM
LIVER DISEASE, ALCOHOLIC
DISEASES ASSOCIATED WITH, 6324
ZIEVES SYNDROME
DISEASES ASSOCIATED WITH, 6319

INTESTINE
SEE ALSO SMALL INTESTINE
ABSORPTION
CHYMOTRYPSIN, 5342*
ALBUMIN
PARASITES AND PARASITIC DISEASE, 6564
CIRCULATION
ECHOGRAPHY, 5585
DISACCHARIDASE
DIETARY FACTORS, 6526
MALABSORPTION, 6526
DISEASES
BILE ACIDS, 6497
DRUG EFFECTS ON
ALKALATING AGENTS, 5584
ATROPINE, 5584
IMMUNOLOGY
PARASITES AND PARASITIC DISEASE, 6565
INFECTION, BACTERIA
VACCINE, 6533

INTESTINE (continued)
LYMPHATICS
OBSTRUCTION, 5586
MOTILITY
CONSTIPATION, 6482*
DIARRHEA, 6482*
GASTROENTERITIS, 6477*
TECHNIQUES, 6477*, 6482*
NEOPLASMS, MALIGNANT
SURGERY, 5612
PARASITES AND PARASITIC DISEASE
EPIDEMIOLOGY, 6567, 6573
THERAPY, 6574
PROTEIN
ABSORPTION, 5266
STOMACH
ACID SECRETION, 5325

INTOLERANCE
CARBOHYDRATE
COMPLICATIONS, 6517
DIARRHEA, 6517
GLUCOSE
HEPATECTOMY, 6096*
JAUNDICE, OBSTRUCTIVE, 5389*
LACTOSE
DIARRHEA, 6491
THERAPY, 6491

INTRINSIC FACTOR
STOMACH
MORPHOLOGY, 5213*

INTUSSUSCEPTION
JEJUNUM
NEOPLASMS, BENIGN, 5868*
SCANNING, SCINTILLATION, 5635*

ION TRANSPORT
SMALL INTESTINE
LAXATIVES, 5269*
SURFACTANTS, 5269*
STOMACH
PEPTIC ULCER, 5260

IRRADIATION
ABDOMEN
SMALL INTESTINE, 5581
GASTROINTESTINAL
NEOPLASMS, MALIGNANT, 5613
HEPATECTOMY
TECHNIQUES, 5510
JEJUNUM
KINETICS, CELL, 5212*, 5589
MORPHOLOGY, 5212*
LIVER
LIVER FUNCTION TESTS, 5509
METASTASES, 6085*
MORPHOLOGY, 5508
LIVER INJURY, 5506
NUCLEIC ACIDS
SYNTHESIS, 5234
PANCREAS
TRYPSIN, 5609
SMALL INTESTINE
ISCHEMIA, 5233
KINETICS, CELL, 5592*
MORPHOLOGY, 5233

IRRADIATION (continued)
SMALL INTESTINE (continued)
PATHOLOGY, 5592*
RADIOPROTECTIVE AGENTS, 5233
VITAMIN B12, 5598*

IRRITABLE COLON
ANTICHOLINERGIC AGENTS, 5982
DRUG TREATMENT, 5916
RADIOLOGY, 6005

ISCHEMIA
COLITIS
ESOPHAGUS, 5985
PATHOLOGY, 5987
RADIOLOGY, 5987
COLON
NEOPLASMS, MALIGNANT, 5965
ILEUM
SCANNING, SCINTILLATION, 5605
LIVER
ANTIENZYMES, 6133
GLYCOPROTEINS, 6133
LIVER FUNCTION TESTS, 5384*
PREVENTION, 5537
REGENERATION, 5384*
SURGERY, 5515
TRANSPLANTATION, 5537
MEGACOLON
DISEASES ASSOCIATED WITH, 5938*
RECTUM
NEOPLASMS, MALIGNANT, 5965
SMALL INTESTINE
IRRADIATION, 5233
PATHOLOGY, 5219

ISOENZYMES
BLOOD
PHOSPHATASE, ALKALINE, 6113*
LIVER DISEASE
PHOSPHATASE, ALKALINE, 6094*
PANCREAS
GENETICS, 5344*
NEOPLASMS, MALIGNANT, 6024*
PHOSPHATASE, ALKALINE
NEOPLASMS, MALIGNANT, 6115*

ISOTHIOCYANATES
CARBOHYDRATE
METABOLISM, 5448
CHOLESTASIS
LIVER, 5448

JAUNDICE
BILIARY DISEASE
REVIEWS, 6429
BILIRUBIN
BINDING, 6214*
SYNTHESIS, 6216
DIAGNOSIS
REVIEWS, 6427
NEONATE
DIAGNOSIS, 6214*
DRUG-INDUCED, 6212*
SCANNING, SCINTILLATION, 5621*

JAUNDICE, OBSTRUCTIVE
ANGIOGRAPHY
TECHNIQUES, 5701
BILE ACIDS
METABOLISM, 6105*
BLOOD
LIPOPROTEINS, 5365*, 6126*, 6421
COPPER
METABOLISM, 5394*
GLUCOSE
INTOLERANCE, 5389*
LIVER
CIRCULATION, 6416
PATHOLOGY, 5380*
PHOSPHORYLATION, OXIDATIVE, 5380*, 5389*

JEJUNUM
SEE ALSO SMALL INTESTINE
ABSORPTION
FATTY ACIDS, 5246*
PEPTIDES, 5239*
WATER, 5246*
ADENOSINE TRIPHOSPHATASE
BILE ACIDS, 5241*
ATROPHY
CELIAC DISEASE, 5921
CIRCULATION
TECHNIQUES, 5599*
CROHNS DISEASE
PATHOLOGY, 6583
HORMONE
HYPERCHLORHYDRIA, 5296*
INTUSSUSCEPTION
SCANNING, SCINTILLATION, 5635*
KINETICS, CELL
ANTINEOPLASTIC AGENTS, 5589
IRRADIATION, 5212*, 5589
MITOSIS
NERVOUS CONTROL, 5201*
MORPHOLOGY
IRRADIATION, 5212*
NEOPLASMS, BENIGN
INTUSSUSCEPTION, 5868*
OBSTRUCTION
ABSORPTION, 5580
GLUCOSE, 5580
SECRETION, 5580
POTASSIUM
TRANSPORT, 5257
TRANSPORT
BILE ACIDS, 5241*, 5248*
CHOLESTEROL, 5248*
SURFACTANTS, 5248*
ULTRASTRUCTURE
DIARRHEA, 6524
VIRUS
GASTROENTERITIS, 6536
VITAMIN B6
DEFICIENCY, 5239*
VITAMIN B12
ABSORPTION, 5902*

KETOGENESIS
LIVER
COENZYMES, 5471

KIDNEY
CIRCULATION
HEPATORENAL SYNDROME, 6546*
HYPERPLASIA
SPLEEN, 5511

KIDNEY DISEASE
GASTROINTESTINAL
SURGERY, 6503
HEPATITIS, CHRONIC
ANTIGEN, AUSTRALIA, 6295*
HEPATITIS, VIRAL
COMPLICATIONS, 6264

KIDNEY TRANSPLANTATION
LIVER FUNCTION TESTS, 6247*
PANCREATITIS
BLEEDING, 6071
COMPLICATIONS, 6071
STOMACH
ACID SECRETION, 5751

KINETICS
BILE ACIDS
METABOLISM, 5409*
SULFOBROMOPHTHALEIN
CIRRHOSIS, 6334*
LIVER DISEASE, 5637*
PORTACAVAL SHUNT, 6334*
TRIGLYCERIDE
METABOLISM, 6380*

KINETICS, CELL
JEJUNUM
ANTINEOPLASTIC AGENTS, 5589
IRRADIATION, 5212*, 5589
SMALL INTESTINE
IRRADIATION, 5592*
LACTATION, 5200*

KININS
BLOOD
DUMPING SYNDROME, 5758

KWASHIORKOR
GLUCOSE
METABOLISM, 6543

LACTASE
SEE ALSO DISACCHARIDASE
CECUM
DIETARY FACTORS, 5573*
SMALL INTESTINE
DIETARY FACTORS, 5573*
LACTOSE, 5573*
PROTEIN, 5573*

LACTASE DEFICIENCY
GLUCOSE
TRANSPORT, 5250*
SMALL INTESTINE
LACTOSE INTOLERANCE, 5920
RADIOLOGY, 5920

LACTATION
SMALL INTESTINE
KINETICS, CELL, 5200*

LACTOFERRIN
DUODENUM
PANCREATITIS, CHRONIC, 6050*
PANCREATITIS, CHRONIC
CALCIFICATION, 6050*

LACTOSE
INTOLERANCE
DIARRHEA, 6491
THERAPY, 6491
SMALL INTESTINE
LACTASE, 5573*

LACTOSE INTOLERANCE
LACTASE DEFICIENCY
SMALL INTESTINE, 5920

LACTOSE TOLERANCE
AGE FACTORS, 5924
DIETARY FACTORS, 5924
ETHNIC FACTORS, 5924

LAMBLIASIS
SEE GIARDIASIS

LAPAROSCOPY
CIRRHOSIS, 5694
HEPATITIS, VIRAL, 6263
PANCREAS
TECHNIQUES, 5628*
PORTAL HYPERTENSION, 5694

LARGE INTESTINE
SEE ALSO INTESTINE, APPENDIX, CECUM,
COLON, RECTUM, ANUS, SIGMOID
AMEBIASIS
DIAGNOSIS, 6558
NECROSIS, 6575
THERAPY, 6505
BACTERIA
CHOLESTEROL, 5928*
POLYPS, 5928*
CROHNS DISEASE
THERAPY, 6505
DISEASES
ENDOSCOPY, 5671
THYROID, 5960
DIVERTICULITIS
REVIEWS, 5968
THERAPY, 6505
DIVERTICULUM
RADIOLOGY, 6005
REVIEWS, 5968
ENDOSCOPY
TECHNIQUES, 5684, 5692
INFLAMMATORY BOWEL DISEASE
THERAPY, 6585
LIPIDS
SYNTHESIS, 5569*
MONOAMINOXIDASE
ULCERATIVE COLITIS, 6016
NEOPLASMS
CHILDREN, 5870
NEOPLASMS, MALIGNANT
ANTIGEN, CARCINOEMBRYONIC, 6013*
CHEMOTHERAPY, 5936*
COMPLICATIONS, 5965
DIETARY FACTORS, 5927*

LARGE INTESTINE (continued)
NEOPLASMS, MALIGNANT (continued)
DISEASES ASSOCIATED WITH, 6004
ETHNIC FACTORS, 5948
IMMUNOLOGY, 5940*
LYMPHATICS, 5940*
METASTASES, 6099*
NUCLEIC ACIDS, 5931*
PATHOLOGY, 5948
RADIOTHERAPY, 5933*
SOCIOECONOMIC FACTORS, 5927*
SURGERY, 5933*, 5936*, 5939*, 5989
SURVIVAL, 5939*
PNEUMATOSIS
ETIOLOGY, 5996
OXYGEN, 5929*
RESPIRATORY SYSTEM, 5996
THERAPY, 5929*
POLYPS
EPIDEMIOLOGY, 5990
FAMILIAL FACTORS, 5928*, 5931*
SURGERY, 5775
PSEUDOTUMOR
CHILDREN, 5870
VOLVULUS
OBSTRUCTION, 5988

LAXATIVES
CHOLESTEROL
METABOLISM, 5543
LIVER INJURY
DRUG-INDUCED, 6272
SMALL INTESTINE
ION TRANSPORT, 5269*
MOTILITY, 5269*

LEAD
CYCLIC ADENOSINE MONOPHOSPHATE
LIVER, 5507

LEISHMANIASIS
TRANSMISSION, 6553

LEUKEMIA
BILIRUBIN
METABOLISM, 6156

LEUKOCYTES
CELIAC DISEASE
IMMUNOLOGY, 5901*
CHEMOTAXIS
LIVER DISEASE, ALCOHOLIC, 6316*
DERMATITIS HERPETIFORMIS
IMMUNOLOGY, 5901*
PHAGOCYTOSIS
APPENDICITIS, 5944
HEPATITIS, VIRAL, 6254

LIPASE
BLOOD
LIVER DISEASE, 6123*, 6147

LIPID STORAGE DISEASES
BILE ACIDS
SYNTHESIS, 6128*
LIVER
TRANSPLANTATION, 6092*

LIPIDS
ABSORPTION
GASTRECTOMY, 5774
VAGOTOMY, 5774
ALBUMIN
SMALL INTESTINE, 5600*
ALCOHOL
OXIDATION, 5557
BILE
CHENODEOXYCHOLIC ACID, 5366*
PHENOBARBITAL, 5442
VITAMIN C, 5371*
ERYTHROCYTE
CHOLESTASIS, 6114*
CIRRHOSIS, 6114*
LIVER DISEASE, 6114*
LIVER
CARBON TETRACHLORIDE, 5516
CHEMICAL COMPOSITION, 5493
DIETARY FACTORS, 5433
HEPATECTOMY, 5532
HYPERLIPEMIA, 6202
PATHOLOGY, 5521
METABOLISM
ALCOHOL, 5414*
ATHEROSCLEROSIS, 5538
CIRRHOSIS, 6301
DIETARY FACTORS, 5383*
FIBER, 5538
HEPATITIS, CHRONIC, 6301
LIPOPROTEINS, 5414*
LIVER, 5383*, 5545
PANCREAS DISEASE, 6043
PERFUSION, 5414*
SMALL INTESTINE, 5568*
TRIGLYCERIDE, 5383*
PEROXIDATION
RADIOPROTECTIVE AGENTS, 5578
SMALL INTESTINE, 5578
SPLEEN
PATHOLOGY, 5521
SYNTHESIS
DIETARY FACTORS, 5569*
LARGE INTESTINE, 5569*
SMALL INTESTINE, 5569*

LIPOPROTEINS
BILE DUCT
OBSTRUCTION, 5365*
BILIARY
ATRESIA, 5624*
BLOOD
CHOLESTASIS, 6126*
HEPATITIS, 5624*
JAUNDICE, OBSTRUCTIVE, 5365*, 6126*, 6421
LIVER DISEASE, 6126*
CHOLESTASIS
DIAGNOSIS, 6181
CHOLESTEROL
SYNTHESIS, 5358*
HEPATITIS
NEONATE, 5624*
LIPIDS
METABOLISM, 5414*
LIVER
ALCOHOLS, POLYHYDRIC, 5430
SYNTHESIS
LIVER, 5430

LIVER
ABSCESS
AMEBIASIS, 6549*, 6551*, 6571
DIAGNOSIS, 6170
DRAINAGE, 5627*
DRUG TREATMENT, 6551*
HEPATECTOMY, 6152
PERFORATION, 6132
RADIOLOGY, 6132
RECURRENCE, 6571
SCANNING, SCINTILLATION, 5627*
SURGERY, 6170
ADENOSINE TRIPHOSPHATASE
HYPOCHOLESTEREMIC AGENTS, 5531
AFLATOXIN
METABOLISM, 5524
ALBUMIN
METABOLISM, 5402*
ALCOHOL
DEHYDROGENASE, 5528
METABOLISM, 5487, 5489
OXIDATION, 5557
ALDEHYDES
DEHYDROGENASE, 5539
AMEBIASIS
DIAGNOSIS, 6549*, 6558
DISEASES ASSOCIATED WITH, 6552
THERAPY, 6158
AMINO ACIDS
CARBON TETRACHLORIDE, 5419*
METABOLISM, 5468, 5476, 5522
SYNTHESIS, 5412*
ANGIOGRAPHY
TECHNIQUES, 5644
ANOMALY, CONGENITAL
CYSTS, 6121*
FIBROSIS, 6121*
ANTIGEN
METABOLISM, 5542
ANTIGEN, AUSTRALIA
CARRIER STATE, 6251, 6286
HEPATITIS, VIRAL, 6251, 6286
ANTITRYPSIN, ALPHA
DEFICIENCY, 6098*
ASCITES
REGENERATION, 6080*
BILE ACIDS
ANALYSIS, 6124*
CHEMICAL COMPOSITION, 5552
LIVER DISEASE, 6124*
METABOLISM, 5388*, 5403*, 5480, 6102*, 6120*
SECRETION, 5353*
SYNTHESIS, 5353*
BILE DUCT
ANOMALY, CONGENITAL, 6109*, 6396*
CYSTS, 6396*
FIBROSIS, 6428
PATHOLOGY, 6168
SHUNT, 6109*
ULTRASTRUCTURE, 5215*
BILIRUBIN
METABOLISM, 5421, 5439
CADMIUM
OCCUPATIONAL FACTORS, 6229
CARBOHYDRATE
METABOLISM, 5448, 5496
CARBON DISULFIDE
METABOLISM, 5491

LIVER (continued)
CARCINOGENS
METABOLISM, 5527
CHOLESTASIS
ETIOLOGY, 5408*, 6135
HEPATITIS, VIRAL, 6240*
ISOTHIOCYANATES, 5448
PREGNANCY, 6108*
REVIEWS, 6427
SURGERY, 6135
ULTRASTRUCTURE, 5408*
CHOLESTEROL
HEPATITIS, VIRAL, 6240*
METABOLISM, 5379*, 5543, 5614
SYNTHESIS, 5358*, 5471, 5529
CIRCULATION
CIRRHOSIS, 6350
GLUCOSE, 5399*
JAUNDICE, OBSTRUCTIVE, 6416
LIVER DISEASE, 5682
LIVER FUNCTION TESTS, 5420
MORPHOLOGY, 5210*
PERFUSION, 5438
PORTAL HYPERTENSION, 6343*
PRIMATES, 5210*
SCANNING, SCINTILLATION, 6350
SECRETIN, 5445
SEPSIS, 5404*
STARVATION, 5404*
SURGERY, 6416
VASOPRESSIN, 5393*, 5415*, 5533
COENZYMES
KETOGENESIS, 5471
COLLAGEN
CIRRHOSIS, 6119*
FIBROSIS, 6119*
COMA
PREVENTION, 6342*
STEROID, 6342*
CONNECTIVE TISSUE
CIRRHOSIS, 6119*
FIBROSIS, 6119*
CONTRAST MEDIA
EXCRETION, 5362*, 5683
COPPER
METABOLISM, 5394*
CRYOSURGERY
GLUCURONIDASE, BETA, 5512
LYSOSOMES, 5512
CYCLIC ADENOSINE MONOPHOSPHATE
ENDOTOXIN, 5507
LEAD, 5507
CYSTS
DISEASES ASSOCIATED WITH, 6419
ECHINOCOCCOSIS, 6570
ECHOGRAPHY, 5636*
HEPATECTOMY, 6152
CYTOCHROMES
ALCOHOL, 5406*
DRUG METABOLISM
ALCOHOL, 5558
ANALGESICS, 5451
ANILINE, 5451
ANTIBACTERIALS, 6218*
ANTICOAGULANTS, 5453
ANTIDIABETIC AGENTS, 6150
ANTINEOPLASTIC AGENTS, 5295
BILIARY DISEASE, 6404*
CIRRHOSIS, 6078*

LIVER (continued)
DRUG METABOLISM (continued)
ENZYMES, 5553
HEPATITIS, CHRONIC, 6310
HYDROCARBONS, 5553
HYDROCARBONS, POLYCYCLIC, 5558, 6150
INSECTICIDES, 5554
LIVER COMA, 6078*
LIVER DISEASE, 6150
PRIMATES, 5295
REVIEWS, 5440
SEX FACTORS, 5558
DRUG TOXICITY, 5548
DYES
EXCRETION, 5547
ECHINOCOCCOSIS
ANGIOGRAPHY, 5677
SURGERY, 6556
ENZYMES
ALCOHOL, 5377*
GLUTATHIONE, 5476
HEPATECTOMY, 5418*
TRANSPLANTATION, 5447
ESTROGENS
METABOLISM, 5459
FATTY ACIDS
DIETARY FACTORS, 5433
METABOLISM, 5363*
OXIDATION, 5551
SYNTHESIS, 5546
FIBROSIS
SCHISTOSOMIASIS, 6003
FOLIC ACID
METABOLISM, 5454, 5482
GLUCONEOGENESIS
ADRENERGIC BLOCKING AGENTS, 5484
FATTY ACIDS, 5363*
HORMONE CONTROL, 5470
SYMPATHOMIMETIC AGENTS, 5484
GLUCOSAMINE
METABOLISM, 5505
GLUCOSE
SYNTHESIS, 5452
GLYCOGEN
HEPATECTOMY, 5532
GLYCOGEN STORAGE DISEASE
FRUCTOSE, 5218
HEMANGIOMA
COMPLICATIONS, 6172
DIAGNOSIS, 6081*
NEONATE, 6081*
SURGERY, 6136, 6172
HEMATOMA
DRAINAGE, 6203
HEME
METABOLISM, 5356*
HYDROCARBONS, HALOGENATED
METABOLISM, 5477
HYPERPLASIA
BLEEDING, 6200
ORAL CONTRACEPTIVES, 6200
HYPERTROPHY
PORTACAVAL SHUNT, 5364*
INSECTICIDES
METABOLISM, 5477, 5530
IRRADIATION
LIVER FUNCTION TESTS, 5509

LIVER (continued)
ISCHEMIA
ANTIENZYMES, 6133
GLYCOPROTEINS, 6133
LIVER FUNCTION TESTS, 5384*
PREVENTION, 5537
REGENERATION, 5384*
SURGERY, 5515
TRANSPLANTATION, 5537
LIPIDS
CARBON TETRACHLORIDE, 5516
CHEMICAL COMPOSITION, 5493
DIETARY FACTORS, 5433
HEPATECTOMY, 5532
HYPERLIPEMIA, 6202
METABOLISM, 5383*, 5545
LIPOPROTEINS
ALCOHOLS, POLYHYDRIC, 5430
SYNTHESIS, 5430
LYSOSOMES
PEPTIDASE, 5494
MERCURY
TOXICITY, 5395*
METABOLISM
REVIEWS, 5463, 6134
METALS
CADMIUM, 5526
METASTASES
BILE ACIDS, 6105*
CIRRHOSIS, 6356
DIAGNOSIS, 5669
ENZYMES, 5669
GASTROINTESTINAL TRACT, 6171
IRRADIATION, 6085*
SURVIVAL, 6099*
MITOCHONDRIA
CARCINOGENS, 5497
GLUTATHIONE, 5497
MITOSIS
HEPATECTOMY, 5532
STARVATION, 5532
MORPHOLOGY
BLOOD SUBSTITUTES, 5223
HEPATECTOMY, 5508
HYDROCARBONS, HALOGENATED, 5223
IRRADIATION, 5508
TRANSPLANTATION, 5447
NECROSIS
ENDOTOXIN, 5397*
TRANSPLANTATION, 6167
NEOPLASMS
CIRRHOSIS, 6106*
SARCOIDOSIS, 6106*
NEOPLASMS, BENIGN
ORAL CONTRACEPTIVES, 6191
PATHOLOGY, 6191
THERAPY, 6107*
NEOPLASMS, MALIGNANT, 6146
ALCOHOLISM, 6195
ANGIOGRAPHY, 5696, 6083*
ANTIGEN, AUSTRALIA, 6138, 6143, 6195, 6236*, 6304, 6357
ANTITRYPSIN, ALPHA, 6087*
CHEMOTHERAPY, 6088*, 6196, 6199, 6206
CHILDREN, 6149
CIRRHOSIS, 6091*
CONTRAST MEDIA, 6201
DIAGNOSIS, 6149, 6209

LIVER (continued)
NEOPLASMS, MALIGNANT (continued)
DISEASES ASSOCIATED WITH, 6091*, 6131, 6142
EPIDEMIOLOGY, 6143
ETHNIC FACTORS, 6143
ETIOLOGY, 6090*
FETOPROTEIN, ALPHA, 5417*, 6131, 6195
GEOGRAPHICAL FACTORS, 6248*
HEPATECTOMY, 6152
HEPATITIS, CHRONIC, 6304
HEPATITIS, VIRAL, 6248*
IMMUNOLOGY, 6149
METASTASES, 6142, 6162
OMENTUM, 6162
PATHOLOGY, 6090*, 6091*, 6142, 6160
PHOSPHATASE, ALKALINE, 6115*
PREVENTION, 6248*
PROTEIN, 5460
RADIOLOGY, 5696
RADIOTHERAPY, 6196, 6199
SCANNING, SCINTILLATION, 5630*, 6083*
SURGERY, 6083*, 6088*, 6151, 6196
SURVIVAL, 6088*
THERAPY, 6131, 6196
NITROSAMINES
BINDING, 5407*
NUCLEIC ACIDS
ANOXIA, 5427
CARCINOGENS, 5458
SYNTHESIS, 5407*, 5490, 5555
OXYGEN
SEPSIS, 5404*
STARVATION, 5404*
PATHOLOGY
ALCOHOL, 5377*
ANTIBIOTICS, 6223*
ANTITRYPSIN, ALPHA, 6098*
CADMIUM, 5369*
CARCINOGENS, 5527
CHOLESTEROL, 5536
CIRCULATION, 6169
CROHNS DISEASE, 6587
FRUCTOSE, 5218
GENETIC FACTORS, 6098*
HEPATITIS, VIRAL, 6240*
HYPERALIMENTATION, 6220*
INFECTION, VIRUS, 6144
JAUNDICE, OBSTRUCTIVE, 5380*
LIPIDS, 5521
MERCURY, 5395*
PELIOSIS HEPATIS, 6176
PORPHYRIA, 6157
REYES SYNDROME, 6095*, 6110*, 6233
WILSONS DISEASE, 6180
PERFUSION
SPECIES DIFFERENCES, 5492
PEROXIDATION
CARBON TETRACHLORIDE, 5419*
PHAGOCYTOSIS
CALCIUM, 5391*
PHOSPHATE
METABOLISM, 5475
PHOSPHOLIPID
CHEMICAL COMPOSITION, 5540
GALACTOSAMINE, 5503

LIVER (continued)
PHOSPHOLIPID (continued)
METABOLISM, 5545
SYNTHESIS, 5472
PHOSPHORYLATION, OXIDATIVE
ANTIBIOTICS, 5443
DRUG EFFECTS ON, 5413*
FOOD ADDITIVES, 5450
JAUNDICE, OBSTRUCTIVE, 5380*, 5389*
SHOCK, 5413*
PORPHYRIA
REVIEWS, 6174
PRECANCER
NITROSAMINES, 5523
VITAMIN C, 5523
PROLAPSE
HERNIA, 6166
PROTEIN
DEFICIENCY, 5456
HORMONE CONTROL, 5426
HYPOXIA, 5437
METABOLISM, 5519, 5542
SECRETION, 5374*
SYNTHESIS, 5372*, 5478, 5479, 5488, 5495, 5522, 5550, 6139
TEMPERATURE, 5437
THYROID, 5426
PSEUDOTUMOR
PATHOLOGY, 6100*
THERAPY, 6107*
REGENERATION
AGE FACTORS, 5534
ENZYMES, 5357*, 5556
FATTY LIVER, 5422
FETOPROTEIN, ALPHA, 5534, 6117*
HUMORAL FACTORS, 5400*
OBESITY, 5422
PERFUSION, 5392*
RIBOSOMES, 5469
SCANNING, SCINTILLATION, 5465
SPLEEN, 5511
TISSUE CULTURE, 5405*
RIBOSOMES
CARCINOGENS, 5458
PHENOBARBITAL, 5479
RUPTURE
AUTOIMMUNITY, 6198
CORTICOSTEROIDS, 6198
DISEASES ASSOCIATED WITH, 6198
SCANNING, SCINTILLATION
BILIARY DISEASE, 5634*
SCLEROSIS
DISEASES ASSOCIATED WITH, 6184
SIALIC ACID
CARBON TETRACHLORIDE, 5419*
STEROID
METABOLISM, 5390*
TRANSPORT, 5520
SULFHYDRYL
OXIDATION, 5464
SULFOBROMOPHTHALEIN
METABOLISM, 5360*
SURGERY
GLUCOSE, 5399*
REGENERATION, 6080*
SEQUELAE, 5399*
TISSUE CULTURE
HEPATITIS, VIRAL, 6279
TRANSPLANTATION
LIPID STORAGE DISEASES, 6092*

LIVER (continued)
TRANSPLANTATION (continued)
LIVER DISEASE, ALCOHOLIC, 6323
PATHOLOGY, 5398*
REJECTION, 5398*
TECHNIQUES, 5498, 5514
TRAUMA
BLEEDING, 6129*
CHILDREN, 6193
DRAINAGE, 6203
HEPATECTOMY, 6152
OPSONIN, 6125*
PHAGOCYTOSIS, 6125*
RETICULOENDOTHELIAL SYSTEM, 6125*
SURGERY, 6129*
TRIGLYCERIDE
FATTY ACIDS, 5435
METABOLISM, 5376*
VITAMIN A, 5549
ULTRASTRUCTURE, 5378*
ALCOHOL, 5513
BURNS, 5502
ENZYMES, 5475
GALACTOSAMINE, 5503
GLYCOPROTEINS, 5449
HEPATECTOMY, 5418*
ISOLATION, 5375*
MAGNESIUM, 5486
PHENOBARBITAL, 5373*
SHOCK, 5501
TISSUE CULTURE, 5449
VITAMIN E, 5504
UREA
DIETARY FACTORS, 5354*
SYNTHESIS, 5354*, 5359*
VIRUS
REYES SYNDROME, 6110*
VITAMIN A
METABOLISM, 5355*
TOXICITY, 5355*
VITAMIN B2
DEFICIENCY, 5525
VITAMIN D
METABOLISM, 5387*

LIVER COMA
AMMONIA
METABOLISM, 5462
BLOOD
PERFUSION, 6239*
DRUG TREATMENT, 5652
ETIOLOGY
SEPSIS, 6112*
IMMUNOGLOBULINS
DEFICIENCY, 6112*
LIVER
DRUG METABOLISM, 6078*
MAGNESIUM
DEFICIENCY, 6161
PROGNOSIS
DRUG METABOLISM, 6078*
THERAPY
REVIEWS, 6367

LIVER DISEASE
ALCOHOLISM
REVIEWS, 6331
ANTIGEN, AUSTRALIA, 6094*

LIVER DISEASE (continued)
ANTITRYPSIN, ALPHA
DEFICIENCY, 6145
AUTOIMMUNITY
ALBUMIN, 6082*
IMMUNOGLOBULINS, 6082*
BILE ACIDS
DIAGNOSIS, 5632*
TOLERANCE, 5620*
BILIARY
SECRETION, 6178
BILIRUBIN
METABOLISM, 6177
BLOOD
BILE ACIDS, 5632*, 6118*, 6124*
FETOPROTEIN, ALPHA, 6117*
FIBRINOLYSIS, 6188
LIPASE, 6123*, 6147
LIPOPROTEINS, 6126*
PHOSPHATASE, ALKALINE, 6113*
PROTEIN, 6137
TRIGLYCERIDE, 6123*
CHILDREN
FETOPROTEIN, ALPHA, 6189
REVIEWS, 6208
CHOLESTEROL
SYNTHESIS, 6194
CORTICOSTEROIDS
METABOLISM, 6097*
DIAGNOSIS
BILE ACIDS, 6118*
INDOCYANINE GREEN, 6079*
SULFOBROMOPHTHALEIN, 5642
DIETARY FACTORS
CARBOHYDRATE, 6084*
DISEASES ASSOCIATED WITH
CIRCULATION, 6130
REVIEWS, 6130
UROGENITAL SYSTEM, 6127*
DRUG TREATMENT, 6207
VITAMIN B12, 6186
ERYTHROCYTE
LIPIDS, 6114*
FETOPROTEIN, ALPHA, 6094*
IMMUNOLOGY, 5700
LIVER
BILE ACIDS, 6124*
CIRCULATION, 5682
DRUG METABOLISM, 6150
LIVER FUNCTION TESTS
SULFOBROMOPHTHALEIN, 6182
METABOLISM
PATHOLOGY, 6104*
REVIEWS, 6104*
NEONATE
REVIEWS, 6208
PANCREAS
SECRETION, 6178
PEPTIC ULCER
DISEASES ASSOCIATED WITH, 5816
PERFUSION
SURVIVAL, 6089*
PHOSPHATASE, ALKALINE
ISOENZYMES, 6094*
PREVENTION
DRUG TREATMENT, 6175
PROTHROMBIN
BLOOD, 6148
REVIEWS, 6134

LIVER DISEASE (continued)
SCANNING, SCINTILLATION
REVIEWS, 5666
TECHNIQUES, 5668, 5699
SMALL INTESTINE
BIOPSY, 5672
SULFOBROMOPHTHALEIN
KINETICS, 5637*
THERAPY
DIET, 6165
REVIEWS, 6141, 6173
TRIGLYCERIDE
METABOLISM, 6123*
ULCERATIVE COLITIS
DISEASES ASSOCIATED WITH, 6012*

LIVER DISEASE, ALCOHOLIC
BLOOD
ESTROGENS, 6317*
CORTICOSTEROIDS
METABOLISM, 6097*
DISEASES ASSOCIATED WITH
INTEGUMENTARY SYSTEM, 6324
DRUG METABOLISM, 6330
EPIDEMIOLOGY, 6272, 6339*
IMMUNOGLOBULINS
DIAGNOSIS, 6320
PROGNOSIS, 6320
IMMUNOLOGY
PROGNOSIS, 6314*
REVIEWS, 6312*, 6328
LEUKOCYTES
CHEMOTAXIS, 6316*
LIVER
TRANSPLANTATION, 6323
PANCREAS
HYALIN, 6052*
PATHOLOGY, 6311*
REVIEWS, 6321, 6327, 6331
SURGERY
REVIEWS, 6322
THERAPY, 6325
TRANSFERRIN
DIAGNOSIS, 6320
PROGNOSIS, 6320
URINE
ACIDITY, 6313*
ESTROGENS, 6317*

LIVER FUNCTION TESTS
BILE ACIDS
TOLERANCE, 5616*
BILIARY DISEASE
SULFOBROMOPHTHALEIN, 6182
BLOOD
METALS, 6140
CHOLESTASIS
DIAGNOSIS, 6126*
CIRRHOSIS, 6337*
DIETARY FACTORS
CARBOHYDRATE, 6084*
DRUG EFFECTS ON
ANTIHORMONES, 5541
FIBRINOLYTIC AGENTS, 6179
IMMUNOSUPPRESSION, 6247*
GASTROINTESTINAL
BLEEDING, 6365
HEPATITIS
VIRUS, 5499

LIVER FUNCTION TESTS (continued)
HEPATITIS, VIRAL
AGE FACTORS, 6281
ANTIGEN, AUSTRALIA, 6274
HYPERALIMENTATION, 6220*
HYPERTENSION
ATHEROSCLEROSIS, 6155
RADIOISOTOPES, 6155
KIDNEY TRANSPLANTATION, 6247*
LIVER
CIRCULATION, 5420
IRRADIATION, 5509
ISCHEMIA, 5384*
LIVER DISEASE
SULFOBROMOPHTHALEIN, 6182
LIVER INJURY
ASPIRIN, 6154
OBESITY
SHUNT, INTESTINAL, 6116*
PREGNANCY
REVIEWS, 6159
REYES SYNDROME, 6110*
SULFOBROMOPHTHALEIN
SCANNING, SCINTILLATION, 6183
TECHNIQUES, 5637*
TECHNIQUES, 5547

LIVER INJURY
ALCOHOL
DRUG TREATMENT, 5548
METABOLISM, 5423
ANALGESICS
REVIEWS, 6231
ASPIRIN
LIVER FUNCTION TESTS, 6154
CARBOHYDRATE
METABOLISM, 5382*
CARBON TETRACHLORIDE, 5535
COENZYMES, 5382*
CYTOCHROMES, 5485
DETERGENTS, 5436
DRUG TREATMENT, 5548
PHOSPHOLIPID, 5419*
THERAPY, 6225*
COMPLICATIONS
NERVOUS SYSTEM, 5444
CYTOCHROMES
AGE FACTORS, 5485
DRUG ADDICTION, 6228
DRUG-INDUCED
ANESTHETICS, 5518, 6232
ANTIARRHYTHMIC AGENTS, 6219*
ANTIBACTERIALS, 6218*
ANTIBIOTICS, 6223*
ANTIINFLAMMATORY AGENTS, 6222*
ANTIMETABOLITES, 5294
GALACTOSAMINE, 5385*
LAXATIVES, 6272
ORAL CONTRACEPTIVES, 6221*
ULTRASTRUCTURE, 5518
ENDOTOXIN
PORTACAVAL SHUNT, 5397*
PORTAL HYPERTENSION, 5397*
GALACTOSAMINE
ALBUMIN, 5446
CLOTTING, 5446
HYDROCARBONS, HALOGENATED
PHENOBARBITAL, 5416*
IRRADIATION, 5506

LIVER INJURY (continued)
NITROSAMINES, 5535
OCCUPATIONAL FACTORS
ECHOGRAPHY, 6224*
SIMULATION
HYPERALIMENTATION, 6220*
VINYL CHLORIDE
ECHOGRAPHY, 6224*

LYMPH
STOMACH
PROTEIN, 5303*

LYMPHANGIECTASIA
COLON, 5961

LYMPHATICS
APPENDIX
MORPHOLOGY, 5203*
INTESTINE
OBSTRUCTION, 5586
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 5940*

LYMPHOCYTE
SMALL INTESTINE
IMMUNOLOGY, 5594*

LYSOSOMES
LIVER
CRYOSURGERY, 5512
PEPTIDASE, 5494
SMALL INTESTINE
PHENOBARBITAL, 5562*

MACROAMYLASE
BLOOD
MALABSORPTION, 5908*

MAGNESIUM
BLOOD
GASTRIN, 5307*
DEFICIENCY
LIVER COMA, 6161
DUODENUM
PANCREATITIS, CHRONIC, 6051*
PARATHYROID GLAND, 6051*
LIVER
ULTRASTRUCTURE, 5486
STOMACH
ACID SECRETION, 5307*

MALABSORPTION
DIAGNOSIS, 5670, 5919
REVIEWS, 5691
TECHNIQUES, 5652
DISACCHARIDE
TOLERANCE TESTS, 5670
DISEASES ASSOCIATED WITH, 5908*
ATHEROSCLEROSIS, 5922
PARASITES AND PARASITIC DISEASE, 6548*
DRUG TREATMENT, 5916
ENTERITIS
INFECTION, VIRUS, 5904*
ETIOLOGY
ENDOCRINE SYSTEM, 5926
MALNUTRITION, 5926

MALABSORPTION (continued)
ETIOLOGY (continued)
PARASITES AND PARASITIC DISEASE, 5926
IMMUNOGLOBULINS
DEFICIENCY, 5903*, 5914
INTESTINE
DISACCHARIDASE, 6526
MACROAMYLASE
BLOOD, 5908*
MENETRIERS DISEASE
DISEASES ASSOCIATED WITH, 5737*
REGIONAL ENTERITIS
DISEASES ASSOCIATED WITH, 6586
SMALL INTESTINE
BIOPSY, 5672
DIVERTICULUM, 5917
REGENERATION, 5913
STOMACH
PROTEIN, 5737*
SURGERY, 5737*
VITAMIN B12
CHILDREN, 5918
GROWTH RETARDATION, 5918
SHUNT, INTESTINAL, 5899

MALIGNANT
SEE NEOPLASMS, MALIGNANT

MALLORY-WEISS SYNDROME
DIAGNOSIS, 5663
SURGERY, 5712
THERAPY, 5712

MALNUTRITION
CIRRHOSIS
AMINO ACIDS, 6366
DRUG TREATMENT, 6366
ETIOLOGY, 6353
GLYCOSIDE
ABSORPTION, 6485*
MALABSORPTION
ETIOLOGY, 5926
PANCREAS
REGENERATION, 6481*
SECRETION, 6481*
SUGAR
TRANSPORT, 6485*

MANOMETRY
SEE PRESSURE STUDIES

MARASMUS
GLUCOSE
METABOLISM, 6543

MECKELS DIVERTICULUM
BLEEDING
SCANNING, SCINTILLATION, 5687
CHILDREN, 5871
COMPLICATIONS
SURGERY, 5860*

MEGACOLON
ANOMALY, CONGENITAL
CHILDREN, 5953
NEONATE, 5953

MEGACOLON (continued)
DISEASES ASSOCIATED WITH
ISCHEMIA, 5938*
ULCERATIVE COLITIS
COMPLICATIONS, 5973

MELENA
COLON
NEOPLASMS, MALIGNANT, 5975

MENETRIERS DISEASE
DISEASES ASSOCIATED WITH
MALABSORPTION, 5737*
STOMACH
POLYPS, 5780

MERCURY
LIVER
PATHOLOGY, 5395*
TOXICITY
LIVER, 5395*

METALS
BLOOD
LIVER FUNCTION TESTS, 6140
OBESITY, 6140
ILEUM
CADMIUM, 5526
LIVER
CADMIUM, 5526

METASTASES
GASTROINTESTINAL TRACT
NEOPLASMS, MALIGNANT, 6171
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 6099*
LIVER
BILE ACIDS, 6105*
CIRRHOSIS, 6356
DIAGNOSIS, 5669
ENZYMES, 5669
GASTROINTESTINAL TRACT, 6171
IRRADIATION, 6085*
NEOPLASMS, MALIGNANT, 6142, 6162
SURVIVAL, 6099*
STOMACH
DISEASES ASSOCIATED WITH, 5735*

MICROORGANISMS
GLYCOSIDE
ABSORPTION, 6485*
SMALL INTESTINE
BILIARY DISEASE, 6411
SUGAR
TRANSPORT, 6485*
ULCERATIVE COLITIS
ILEOSTOMY, 6022

MITOCHONDRIA
ALCOHOL
OXIDATION, 5557
LIVER
CARCINOGENS, 5497
GLUTATHIONE, 5497
SMALL INTESTINE
MORPHOLOGY, 5202*

MITOSIS
GASTROINTESTIMAL TRACT
HYPERALIMENTATION, 5595*
JEJUNUM
NERVOUS CONTROL, 5201*
LIVER
HEPATECTOMY, 5532
STARVATION, 5532
SMALL INTESTINE
MORPHOLOGY, 5202*

MONOAMINOXIDASE
LARGE INTESTINE
ULCERATIVE COLITIS, 6016

MORPHINE
SMALL INTESTINE
MOTILITY, 5207*

MORTALITY
SEE SURVIVAL

MOTILIN
BLOOD
RADIOIMMUNOASSAY, 5273*
DUODENUM
ACIDITY, 5273*

MOTILITY
COLON
BILE ACIDS, 5245*
CALCIUM, 5268*
ELECTROLYTE, 5268*
NERVOUS CONTROL, 5270*
DUODENUM
GLUCAGON, 5626*
ESOPHAGUS
ANTIDIARRHEALS, 5277
TRYPANOSOMIASIS, 6547*
GALLBLADDER
ANTIEMETICS, 6467
GASTRECTOMY, 5749
RADIOLOGY, 6467
VAGOTOMY, 5749
GASTROINTESTINAL
ALCOHOL, 5254
CHOLERETIC AGENTS, 5289
DRUG EFFECTS ON, 5254, 5652
HORMONE CONTROL, 5279
NERVOUS CONTROL, 5278
REVIEWS, 5279
ILEUM
CALCIUM, 5268*, 5283
DRUG-INDUCED, 5283
ELECTROLYTE, 5268*
SODIUM, 5283
INTESTINE
CONSTIPATION, 6482*
DIARRHEA, 6482*
GASTROENTERITIS, 6477*
TECHNIQUES, 6477*, 6482*
SMALL INTESTINE
ACETYLCHOLINE, 5267*
ADRENERGIC AGENTS, 5267*
H2 RECEPTOR ANTAGONISTS, 5564*
HISTAMINE, 5274*, 5564*
LAXATIVES, 5269*
MORPHINE, 5267*
NERVOUS SYSTEM, 5274*
SURFACTANTS, 5269*

MOTILITY (continued)
STOMACH
ALKALOIDS, 5284
ANTIEMETICS, 5738*
CALCITONIN, 5769
CHOLECYSTOKININ, 5271*
CHOLINESTERASE, 5272*
DRUG EFFECTS ON, 5842
DUODENUM, 5275*
HYPERCHLORHYDRIA, 5769
MARKER STUDIES, 5275*
NERVOUS CONTROL, 5272*
OSMOTIC PRESSURE, 5275*
PEPTIC ULCER, 5823
PROSTAGLANDIN, 5281
REVIEWS, 5282
SEROTONIN, 5284
TRACER STUDY, 5752, 5823

MOUTH
CROHNS DISEASE, 6582*
ULCERATIVE COLITIS, 6582*

MUCOPOLYSACCHARIDE
ENZYMES
COLON, 5211*
HYDROLYSIS
COLON, 5211*
SMALL INTESTINE
WHIPPLES DISEASE, 5915

MUCUS
GALLBLADDER
ULTRASTRUCTURE, 5236

MUSCULOSKELETAL SYSTEM
ABDOMEN
ANOMALY, CONGENITAL, 6496

MYCOSIS
SEE INFECTION, FUNGUS

NECROSIS
COLON
AMEBIASIS, 5992
ETIOLOGY, 5972
SURGERY, 5972
LARGE INTESTINE
AMEBIASIS, 6575
LIVER
ENDOTOXIN, 5397*
TRANSPLANTATION, 6167
SMALL INTESTINE
AMEBIASIS, 6575
DISEASES ASSOCIATED WITH, 6552
TRANSPLANTATION, 5577

NEONATE
SEE ALSO AGE FACTORS, CHILDREN
DIARRHEA
DRUG TREATMENT, 6490
GASTROENTERITIS
THERAPY, 6507
HEPATITIS
LIPOPROTEINS, 5624*
PATHOLOGY, 6211*
PROGNOSIS, 6211*
HIRSCHSPRUNGS DISEASE
RADIOLOGY, 5623*

NEONATE (continued)
JAUNDICE
DIAGNOSIS, 6214*
DRUG-INDUCED, 6212*
SCANNING, SCINTILLATION, 5621*
LIVER
HEMANGIOMA, 6081*
LIVER DISEASE
REVIEWS, 6208
MEGACOLON
ANOMALY, CONGENITAL, 5953
PROTEIN
ABSORPTION, 5266
PYLORUS
STENOSIS, 5791
WHIPPLES DISEASE
ETIOLOGY, 5911

NEOPLASMS
APPENDIX
PATHOLOGY, 5977
COLON
PARASITES AND PARASITIC DISEASE, 6554
LARGE INTESTINE
CHILDREN, 5870
LIVER
CIRRHOSIS, 6106*
SARCOIDOSIS, 6106*
PANCREAS
GASTRIN, 5593*, 6483*
REVIEWS, 6515
SURGERY, 6515
TISSUE CULTURE, 5593*
ZOLLINGER-ELLISON SYNDROME, 5593*
SALIVARY GLANDS
ULTRASTRUCTURE, 6495
SMALL INTESTINE
CHILDREN, 5870
RADIOLOGY, 5656
ZOLLINGER-ELLISON SYNDROME
REVIEWS, 6515

NEOPLASMS, BENIGN
SEE ALSO POLYPS
ANUS
TRANSFORMATION, 5976
BILIARY
SURGERY, 6448
GALLBLADDER
DISEASES ASSOCIATED WITH, 6464
JEJUNUM
INTUSSUSCEPTION, 5868*
LIVER
ORAL CONTRACEPTIVES, 6191
PATHOLOGY, 6191
THERAPY, 6107*
SMALL INTESTINE
ANGIOGRAPHY, 5655
STOMACH
BIOPSY, 5615*
DIAGNOSIS, 5766
ENDOSCOPY, 5615*
SURGERY, 5766, 5773, 5777
ULTRASTRUCTURE, 5781

NEOPLASMS, MALIGNANT
AMPULLA OF VATER, 6466
PANCREATECTOMY, 6519

NEOPLASMS, MALIGNANT (continued)
ANUS
SURGERY, 5969
BILE DUCT
CYTOLOGY, 5631*
DISEASES ASSOCIATED WITH, 6419
SURGERY, 6432, 6435, 6445
BILIARY
AFLATOXIN, 5483
PRIMATES, 5483
CARCINOIDS
GASTROINTESTINAL, 6487
CECUM
RUPTURE, 5998
CELIAC DISEASE
DISEASES ASSOCIATED WITH, 5921
COLON, 5868*
ANTIGEN, CARCINOEMBRYONIC, 5935*
CECUM, 5998
DIAGNOSIS, 5981
ENDOSCOPY, 5692
ISCHEMIA, 5965
MELENA, 5975
PATHOLOGY, 5978
PERFORATION, 5975
RADIOLOGY, 5932*
SURGERY, 5974, 5981
SURVIVAL, 6000
DUODENUM
CHEMOTHERAPY, 6484*
DIAGNOSIS, 6484*
ENDOSCOPY, 5897
ESOPHAGUS
DIAGNOSIS, 5709
DISEASES ASSOCIATED WITH, 5705*, 5717, 5732
EPIDEMIOLOGY, 5703*, 5727
HETEROTOPIA, 5705*
PATHOLOGY, 5710
RADIOTHERAPY, 5710, 5715
SURGERY, 5711, 5720, 5731
FISTULA
RECTUM, 5950
GALLBLADDER, 6423
PATHOLOGY, 6399*
SURGERY, 6399*, 6407
SURVIVAL, 6407
GASTROINTESTINAL
ANTIGEN, CARCINOEMBRYONIC, 5688
COMPLICATIONS, 6516
ENDOSCOPY, 5641
IMMUNOLOGY, 6498
IRRADIATION, 5613
NITROSAMINES, 5613
PERFORATION, 6516
RADIOTHERAPY, 6499
GASTROINTESTINAL TRACT
METASTASES, 6171
INTESTINE
SURGERY, 5612
LARGE INTESTINE
ANTIGEN, CARCINOEMBRYONIC, 6013*
CHEMOTHERAPY, 5936*
COMPLICATIONS, 5965
DIETARY FACTORS, 5927*
DISEASES ASSOCIATED WITH, 6004
ETHNIC FACTORS, 5948
IMMUNOLOGY, 5940*
LYMPHATICS, 5940*

NEOPLASMS, MALIGNANT (continued)
LARGE INTESTINE (continued)
METASTASES, 6099*
NUCLEIC ACIDS, 5931*
PATHOLOGY, 5948
RADIOTHERAPY, 5933*
SOCIOECONOMIC FACTORS, 5927*
SURGERY, 5933*, 5936*, 5939*, 5989
SURVIVAL, 5939*
LIVER, 6146
ALCOHOLISM, 6195
ANGIOGRAPHY, 5696, 6083*
ANTIGEN, AUSTRALIA, 6138, 6143, 6195, 6236*, 6304, 6357
ANTITRYPSIN, ALPHA, 6087*
CHEMOTHERAPY, 6088*, 6196, 6199, 6206
CHILDREN, 6149
CIRRHOSIS, 6091*
CONTRAST MEDIA, 6201
DIAGNOSIS, 6149, 6209
DISEASES ASSOCIATED WITH, 6091*, 6131, 6142
EPIDEMIOLOGY, 6143
ETHNIC FACTORS, 6143
ETIOLOGY, 6090*
FETOPROTEIN, ALPHA, 5417*, 6131, 6195
GEOGRAPHICAL FACTORS, 6248*
HEPATECTOMY, 6152
HEPATITIS, CHRONIC, 6304
HEPATITIS, VIRAL, 6248*
IMMUNOLOGY, 6149
METASTASES, 6142, 6162
OMENTUM, 6162
PATHOLOGY, 6090*, 6091*, 6142, 6160
PHOSPHATASE, ALKALINE, 6115*
PREVENTION, 6248*
PROTEIN, 5460
RADIOLOGY, 5696
RADIOTHERAPY, 6196, 6199
SCANNING, SCINTILLATION, 5630*, 6083*
SURGERY, 6083*, 6088*, 6151, 6196
SURVIVAL, 6088*
THERAPY, 6131, 6196
NUCLEIC ACIDS
SYNTHESIS, 5931*
PANCREAS
ANGIOGRAPHY, 5639*
ANTIGEN, CARCINOEMBRYONIC, 6033
CLOTTING, 6037
CYTOLOGY, 5631*
DIAGNOSIS, 5690
DISEASES ASSOCIATED WITH, 6025*, 6034, 6042
FETOPROTEIN, ALPHA, 6033
HUMORAL FACTORS, 6028*
IMMUNOLOGY, 6033
ISOENZYMES, 6024*
PATHOLOGY, 6028*, 6031
PEPTIC ULCER, 5847
PHOSPHATASE, ALKALINE, 6024*
RADIOLOGY, 5689, 5693
SCANNING, SCINTILLATION, 5665
SECRETIN, 5690
SURGERY, 6037, 6494
TRACER STUDY, 5629*

NEOPLASMS, MALIGNANT (continued)
PAPILLA OF VATER
SURGERY, 6397*
PHOSPHATASE, ALKALINE
ISOENZYMES, 6115*
PYLORUS
STENOSIS, 5771
RECTUM
DIAGNOSIS, 5981
DISEASES ASSOCIATED WITH, 6004
ENDOSCOPY, 5692
ISCHEMIA, 5965
RADIOLOGY, 5932*
RADIOTHERAPY, 5941, 5942
SURGERY, 5941, 5942, 5949, 5981
SURVIVAL, 6000
SALIVARY GLANDS
DISEASES ASSOCIATED WITH, 6535
SMALL INTESTINE
ANGIOGRAPHY, 5655
COMPLICATIONS, 5878
IMMUNOLOGY, 5587
RADIATION, 5588
RADIOLOGY, 5883
STOMACH, 5789
ALBUMIN, 5770
CALCIFICATION, 5778
CHEMOTHERAPY, 6484*
CIRCULATION, 5757
COMPLICATIONS, 5783
DIAGNOSIS, 5651, 5659, 5709, 5756, 6484*
ENDOSCOPY, 5619*, 5641, 5651, 5740, 5763
ENZYMES, 5767
FOREIGN BODIES, 5783
GASTRECTOMY, 5745
IMMUNOGLOBULINS, 5795
MARKER STUDIES, 5619*
NITROSAMINES, 5316
PATHOLOGY, 5736*, 5753, 5757, 5788, 5948
POLYPS, 5780
RADIOLOGY, 5661, 5740
RADIOTHERAPY, 5746, 5750
RECURRENCE, 5792
RESPIRATORY SYSTEM, 5762
REVIEWS, 5796
SIMULATION, 5316
SOCIOECONOMIC FACTORS, 5927*
SURGERY, 5750, 5782, 5786, 5792
SURVIVAL, 5763
THERAPY, 5753
ULCERATIVE COLITIS
ANTIGEN, CARCINOEMBRYONIC, 6013*
COMPLICATIONS, 6015*

NERVOUS CONTROL
ACETYLCHOLINE
SYNTHESIS, 5291
BILE
SPHINCTER, 5517
COLON
MOTILITY, 5276*
GASTROINTESTINAL
MOTILITY, 5278
JEJUNUM
MITOSIS, 5201*

NERVOUS CONTROL (continued)
PANCREAS
SECRETION, 5287*, 5345
STOMACH
ACID SECRETION, 5287*
MOTILITY, 5272*

NERVOUS SYSTEM
BIOPOTENTIALS
CHOLECYSTOKININ, 5603
CIRRHOSIS, 6337*
DISEASES ASSOCIATED WITH, 6370
DRUG TREATMENT, 6335*
ESOPHAGUS
BIOPOTENTIALS, 5280
GASTRITIS
PATHOLOGY, 5747
LIVER INJURY
COMPLICATIONS, 5444
PANCREAS
MORPHOLOGY, 5345
SMALL INTESTINE
HYPERTROPHY, 5571*
MOTILITY, 5274*

NITROGEN
CHYME
FEEDING, 5290
SMALL INTESTINE, 5290
STOMACH, 5290

NITROSAMINES
BINDING
LIVER, 5407*
GASTROINTESTINAL
NEOPLASMS, MALIGNANT, 5613
LIVER
PRECANCER, 5523
LIVER INJURY, 5535
NUCLEIC ACIDS
SYNTHESIS, 5407*
STOMACH
NEOPLASMS, MALIGNANT, 5516

NUCLEIC ACIDS
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 5931*
LIVER
ANOXIA, 5427
CARCINOGENS, 5458
SYNTHESIS
ANTIGEN, AUSTRALIA, 6244*
CIRRHOSIS, 6244*, 6292*
CORTICOSTEROIDS, 5490
CYCLIC ADENOSINE MONOPHOSPHATE, 5555
HEPATECTOMY, 5357*, 5392*, 5556
HEPATITIS, CHRONIC, 6244*, 6292*
HEPATITIS, VIRAL, 6244*
INFECTION, BACTERIA, 5490
IRRADIATION, 5234
LIVER, 5407*, 5490, 5555
NEOPLASMS, MALIGNANT, 5931*
NITROSAMINES, 5407*
SMALL INTESTINE, 5234

NUCLEOTIDASE
GASTROINTESTINAL, 5292

NUCLEOTIDES
ABSORPTION
SMALL INTESTINE, 5238*

OBESITY
BLOOD
METALS, 6140
LIVER
REGENERATION, 5422
SHUNT, INTESTINAL
CHOLELITHIASIS, 6116*
CIRRHOSIS, 6111*
COMPLICATIONS, 6056*
FATTY LIVER, 5570*, 6111*, 6116*
LIVER FUNCTION TESTS, 6116*
PANCREATITIS, 6056*
SURGERY
SHUNT, INTESTINAL, 5894, 5896, 5899, 5900, 6390*, 6403*
TECHNIQUES, 5900

OBSTRUCTION
BILE DUCT
LIPOPROTEINS, 5365*
BILIARY
INFECTION, VIRUS, 5401*
SIMULATION, 5401*
DUODENUM
BILIARY DISEASE, 5891
ILEUM
ENDOSCOPY, 5889
THERAPY, 5889
INTESTINE
LYMPHATICS, 5586
JEJUNUM
ABSORPTION, 5580
GLUCOSE, 5580
SECRETION, 5580
LARGE INTESTINE
VOLVULUS, 5988
SMALL INTESTINE, 5881
DIVERTICULUM, 5869
ENDOMETRIOSIS, 5862*
THERAPY, 5884

OBSTRUCTIVE JAUNDICE
SEE JAUNDICE, OBSTRUCTIVE

OCCUPATIONAL FACTORS
CADMIUM
LIVER, 6229
LIVER INJURY
ECHOGRAPHY, 6224*

OMENTUM
LIVER
NEOPLASMS, MALIGNANT, 6162

OPSONIN
CHOLESTEROL
METABOLISM, 5614
LIVER
TRAUMA, 6125*

ORAL CONTRACEPTIVES
BUDD-CHIARI SYNDROME
ETIOLOGY, 6369

ORAL CONTRACEPTIVES (continued)
LIVER
HYPERPLASIA, 6200
NEOPLASMS, BENIGN, 6191
LIVER INJURY
DRUG-INDUCED, 6221*

OSMOTIC PRESSURE
STOMACH
MOTILITY, 5275*

OXIDATION
ALCOHOL
LIPIDS, 5557
LIVER, 5557
MITOCHONDRIA, 5557
FATTY ACIDS
LIVER, 5551
SULFHYDRYL
LIVER, 5464

OXYGEN
BLOOD
CIRRHOSIS, 6345*
LARGE INTESTINE
PNEUMATOSIS, 5929*
LIVER
SEPSIS, 5404*
STARVATION, 5404*
PEPTIC ULCER
DRUG TREATMENT, 5832
STOMACH
PERMEABILITY, 5265

PAIN
ABDOMEN
SEDATIVES, 5882

PANCREAS
AMYLASE
GENETICS, 5344*
SECRETION, 5350
ARTERY
ANOMALY, 5606
BIOPSY
TECHNIQUES, 5628*
CALCULI
AMYLASE, 6027*
BILE, 6027*
GLUTAMYL TRANSPEPTIDASE, GAMMA, 6027*
SECRETION, 6027*
CHYMOTRYPSIN
SECRETION, 5341*
CIRCULATION
CHILDREN, 5217
GASTROENTEROSTOMY, 6030*
CYSTS
ANGIOGRAPHY, 6040
SURGERY, 6032
CYTOTOXICITY
ALKALOIDS, 5208*
ECHOGRAPHY, 5702
ENDOSCOPY
GLUCAGON, 5626*
ENZYMES
ABSORPTION, 5342*

PANCREAS (continued)
GASTRIN
AGE FACTORS, 5206*
EMBRYOLOGY, 5206*
HETEROTOPIA, 6038
HYALIN
ALCOHOLISM, 6052*
LIVER DISEASE, ALCOHOLIC, 6052*
ISOENZYMES
GENETICS, 5344*
LAPAROSCOPY
TECHNIQUES, 5628*
MORPHOLOGY
DRUG EFFECTS ON, 5338*
NERVOUS SYSTEM, 5345
NEOPLASMS
GASTRIN, 5593*, 6483*
REVIEWS, 6515
SURGERY, 6515
TISSUE CULTURE, 5593*
ZOLLINGER-ELLISON SYNDROME, 5593*
NEOPLASMS, MALIGNANT
ANGIOGRAPHY, 5639*
ANTIGEN, CARCINOEMBRYONIC, 6033
CLOTTING, 6037
CYTOLOGY, 5631*
DIAGNOSIS, 5690
DISEASES ASSOCIATED WITH, 6025*, 6034, 6042
FETOPROTEIN, ALPHA, 6033
HUMORAL FACTORS, 6028*
IMMUNOLOGY, 6033
ISOENZYMES, 6024*
PATHOLOGY, 6028*, 6031
PEPTIC ULCER, 5847
PHOSPHATASE, ALKALINE, 6024*
RADIOLOGY, 5689, 5693
SCANNING, SCINTILLATION, 5665
SECRETIN, 5690
SURGERY, 6037, 6494
TRACER STUDY, 5629*
PATHOLOGY
ANTIMETABOLITES, 5294
ANTINEOPLASTIC AGENTS, 5208*
PEPTIC ULCER
DISEASES ASSOCIATED WITH, 5847
PHOSPHOLIPID
METABOLISM, 5350
PHOSPHORYLATION, OXIDAITVE
PANCREATITIS, 6046*
PROTEIN
SECRETION, 5341*, 5352
SYNTHESIS, 5351
PSEUDOCYST
ABSCESS, 6044
BLEEDING, 6036, 6066
COMPLICATIONS, 6058, 6066
THERAPY, 6058
RADIOLOGY, 6392*
REGENERATION
MALNUTRITION, 6481*
SECRETION
ALCOHOL, 5287*, 5348
ANEMIA, PERNICIOUS, 6029*
ANTIBIOTICS, 5343*
CALCIUM, 5337*
CHOLECYSTOKININ, 5574*
CHOLELITHIASIS, 6391*
CHOLINERGIC AGENTS, 5337*

PANCREAS (continued)
SECRETION (continued)
DRUG EFFECTS ON, 5338*, 6178
ENTERECTOMY, 5425
GASTRECTOMY, 5806
GASTRITIS, ATROPHIC, 6029*
HEMOCHROMATOSIS, 6041
HORMONE EFFECTS ON, 5333
IMMUNOGLOBULINS, 6026*
LIVER DISEASE, 6178
MALNUTRITION, 6481*
NERVOUS CONTROL, 5287*, 5345
PROTEIN, 5349
SECRETIN, 5347
SYMPATHOMIMETIC AGENTS, 5347
ZOLLINGER-ELLISON SYNDROME, 6029*
STOMACH
ACID SECRETION, 5325
TRANSPLANTATION
CYTOLOGY, 5346
GENETICS, 5346
TRAUMA
IMMUNOGLOBULINS, 6026*
TRYPSIN
ANTINEOPLASTIC AGENTS, 5609
GIARDIASIS, 6544*
IRRADIATION, 5609

PANCREAS DISEASE
ANGIOGRAPHY, 6039
REVIEWS, 6383*
TECHNIQUES, 5645, 5646
BLOOD
IMMUNOGLOBULINS, 6055*
DIAGNOSIS
PANCREOZYMIN, 5681
SECRETIN, 5681
TECHNIQUES, 5664
DISEASES ASSOCIATED WITH
BILIARY DISEASE, 6383*
GASTROENTEROSTOMY
SEQUELAE, 6030*
LIPIDS
METABOLISM, 6043
PEPTIC ULCER
DISEASES ASSOCIATED WITH, 5816, 5833
SCANNING, SCINTILLAITON, 5673
REVIEWS, 5666
SMALL INTESTINE
BIOPSY, 5672
STOMACH
VARICES, 6035
SURGERY
TECHNIQUES, 6469

PANCREAS FUNCTION TESTS
CERULEIN, 5657
GASTROENTEROSTOMY, 6048*
SECRETIN
CHOLELITHIASIS, 6391*

PANCREATECTOMY
AMPULLA OF VATER
NEOPLASMS, MALIGNANT, 6519

PANCREATITIS
ALCOHOL
ETIOLOGY, 6049*
TRIGLYCERIDE, 6049*

PANCREATITIS (continued)
BILIARY
SURGERY, 6077
BLEEDING
AMINO ACIDS, 5340*
KIDNEY TRANSPLANTATION, 6071
PROTEASE, 6064
BLOOD
CALCIUM, 5339*
CLOTTING, 6070
IMMUNOGLOBULINS, 6055*
SEROTONIN, 6074
CHOLINE
DEFICIENCY, 5340*
COLON
STENOSIS, 6061
COMPLICATIONS
AGE FACTORS, 6062
KIDNEY TRANSPLANTATION, 6071
SURGERY, 6060
THYROID, 5339*
DIAGNOSIS
CERULEIN, 5657
PERITONEUM, 6047*
TEMPERATURE, 5648
DISEASES ASSOCIATED WITH
AFFERENT LOOP SYNDROME, 6067
CIRCULATION, 6070
HYPERLIPOPROTEINEMIA, 6057*
UROGENITAL SYSTEM, 6067
DRUG-INDUCED
PATHOLOGY, 5229
DRUG TREATMENT
ANTIENZYMES, 6065, 6073
PHOSPHOLIPASE, 6046*
ETIOLOGY
AGE FACTORS, 6062
HYPERLIPOPROTEINEMIA, 6057*
FAMILIAL FACTORS
AMINO ACIDS, 6045*
GASTROINTESTINAL
SURGERY, 6077
OBESITY
SHUNT, INTESTINAL, 6056*
PANCREAS
PHOSPHORYLATION, OXIDAITVE, 6046*
PATHOLOGY, 6072
PREVENTION
GLUCAGON, 5626*
SCANNING, SCINTILLATION, 5665
STOMACH
VARICES, 6035
SURGERY, 6063, 6075
THERAPY, 6063
URINE
AMINO ACIDS, 6045*

PANCREATITIS, CHRONIC
ANGIOGRAPHY, 5639*
CALCIFICATION
IMMUNOLOGY, 6053*
LACTOFERRIN, 6050*
PATHOLOGY, 6054*
SECRETION, 6054*
STOMACH, 6059
CHOLELITHIASIS
DISEASES ASSOCIATED WITH, 6377*, 6391*

PANCREATITIS, CHRONIC (continued)
DIAGNOSIS
SECRETIN, 5647
TECHNIQUES, 6076
TRACER STUDY, 5629*
DISEASES ASSOCIATED WITH, 6042
DUODENUM
CALCIUM, 6051*
LACTOFERRIN, 6050*
MAGNESIUM, 6051*
PATHOLOGY, 6069
ETIOLOGY
CHOLELITHIASIS, 6377*
PANCREOZYMIN
RADIOLOGY, 5693
SECRETIN, 5647
STARCHES
HYDROLYSIS, 5865*
STOMACH
ACID SECRETION, 6059
SURGERY
TECHNIQUES, 6068

PANCREOZYMIN
PANCREAS DISEASE
DIAGNOSIS, 5681
PANCREATITIS, CHRONIC
SECRETIN, 5647

PANETH CELL
SECRETION
DRUG EFFECTS ON, 5576*
STARVATION, 5576*
SMALL INTESTINE
DRUG EFFECTS ON, 5197*

PAPILLA OF VATER
ANGIOGRAPHY, 5676
DISEASES, 6408
SURGERY, 6409
ENDOSCOPY, 5676
NEOPLASMS, MALIGNANT
SURGERY, 6397*
SCANNING, SCINTILLATION, 5676
STENOSIS
DIAGNOSIS, 5640
SURGERY
CHOLEDOCHOLITHIASIS, 6400*
SEQUELAE, 6410

PARASITES AND PARASITIC DISEASE
COLON
NEOPLASMS, 6554
DISEASES ASSOCIATED WITH
ANEMIA, 6562
DRUG TREATMENT, 6545*, 6561, 6562
EPIDEMIOLOGY
CHILDREN, 6572
GASTROINTESTINAL
COMPLICATIONS, 6559
INTESTINE
ALBUMIN, 6564
EPIDEMIOLOGY, 6567, 6573
IMMUNOLOGY, 6565
THERAPY, 6574
MALABSORPTION
DISEASES ASSOCIATED WITH, 6548*
ETIOLOGY, 5926
MORPHOLOGY, 5235

PARASITES AND PARASITIC DISEASE (continued)
THERAPY, 6546*
TRANSMISSION
PREVENTION, 6568
ULTRASTRUCTURE
PHOSPHATASE, ACID, 6563

PARASYMPATHOMIMETIC AGENTS
STOMACH
ACID SECRETION, 5298*

PARATHYROID GLAND
DUODENUM
CALCIUM, 6051*
MAGNESIUM, 6051*

PARATYPHOID FEVER
SEE SALMONELLOSIS

PELIOSIS HEPATIS
ETIOLOGY, 6176
LIVER
PATHOLOGY, 6176

PENTAGASTRIN
STOMACH
ADENYL CYCLASE, 5301*
SECRETION, 5327

PEPSIN
SECRETION
PEPTIC ULCER, 5848
STOMACH, 5848

PEPSINOGEN
URINE
PEPTIC ULCER, 5819

PEPTIC ULCER
SEE ALSO ULCER, STOMACH, DUODENUM
ADOLESCENTS
EPIDEMIOLOGY, 5811
BILE
SECRETION, 5820
BLOOD
GASTRIN, 5804
CHILDREN
EPIDEMIOLOGY, 5811
CIRCULATION, 5830
COMPLICATIONS
ENDOSCOPY, 5828
SURGERY, 5846
DISEASES ASSOCIATED WITH
BILIARY DISEASE, 5816
CIRCULATION, 5837
LIVER DISEASE, 5816
PANCREAS, 5847
PANCREAS DISEASE, 5816, 5833
DRAINAGE
VAGOTOMY, 5850
DRUG TREATMENT, 5805, 5818, 5834, 5858
ANTICHOLINERGIC AGENTS, 5821
CARBENOXALONE, 5799*
COMPLICATIONS, 5799*
OXYGEN, 5832
TERPENES, 5843
ETIOLOGY, 5808, 5852
REFLUX, 5822

PEPTIC ULCER (continued)
FECES
CHEMICAL COMPOSITION, 581.
FOREIGN BODIES
STOMACH, 5813
GASTRIN
BLOOD, 5838, 5839
STOMACH, 5748
HEARTBURN
DISEASES ASSOCIATED WITH, 5831
HYPERCHLORHYDRIA
DISEASES ASSOCIATED WITH, 5831
PANCREAS
NEOPLASMS, MALIGNANT, 5847
PATHOLOGY, 5808
PEPSIN
SECRETION, 5848
PEPSINOGEN
URINE, 5819
PERFORATION
AMYLASE, 585+
COMPLICATIONS, 5755
SURGERY, 5857
PORTACAVAL SHUNT
SEQUELAE, 5288*
PREVENTION
HORMONE, 5333
VAGOTOMY, 5288*
PYLOROPLASTY, 5841
PYLORUS
ANOMALY, 5755
RADIOTHERAPY, 5803
RECURRENCE
SURGERY, 5856
SMALL INTESTINE
DIVERTICULUM, 5875
STOMACH
ACID SECRETION, 5807
ACIDITY, 5825
BIOPOTENTIALS, 5260
DIVERTICULUM, 5875
ION TRANSPORT, 5260
MOTILITY, 5823
PERMEABILITY, 5803
SECRETION, 5800*, 5801*, 5809, 5812, 5819, 5829, 6489
SURGERY
COMPLICATIONS, 5844
REFLUX, 5802*
SEQUELAE, 5802*, 5807, 5836
TECHNIQUES, 5845
VAGOTOMY, 5817
VITAMIN C, 5840
VAGOTOMY, 5841
SEQUELAE, 5802*, 5815, 5824
TECHNIQUES, 5314, 5853

PEPTIDASE
LIVER
LYSOSOMES, 5494
SMALL INTESTINE
DRUG EFFECTS ON, 5197*

PEPTIDES
ABSORPTION
SMALL INTESTINE, 5244*
BILE, 5429
COLON
ULTRASTRUCTURE, 5567*

PEPTIDES (continued)
GASTROINTESTINAL
BIOPOTENTIALS, 5270*
JEJUNUM
ABSORPTION, 5239*
SECRETION
SMALL INTESTINE, 5198*
SMALL INTESTINE
ULTRASTRUCTURE, 5567*
TRANSPORT
SMALL INTESTINE, 5240*

PERFORATION
COLON
AMEBIASIS, 5992
NEOPLASMS, MALIGNANT, 5975
SCHISTOSOMIASIS, 5983
SURGERY, 5955, 6002
ESOPHAGUS
DISEASES ASSOCIATED WITH, 6522
GALLBLADDER
ANGIOGRAPHY, 6452
COMPLICATIONS, 6452
SURGERY, 6475
GASTROINTESTINAL
NEOPLASMS, MALIGNANT, 6516
ILEUM
DISEASES ASSOCIATED WITH, 6522
LIVER
ABSCESS, 6132
PEPTIC ULCER
AMYLASE, 5854
COMPLICATIONS, 5755
SURGERY, 5857

PERFUSION
BLOOD
HEPATITIS, VIRAL, 6239*
LIVER COMA, 6239*
ILEUM
ABSORPTION, 5249*
LIPIDS
METABOLISM, 5414*
LIVER
CIRCULATION, 5438
REGENERATION, 5392*
SPECIES DIFFERENCES, 5492
LIVER DISEASE
SURVIVAL, 6089*
SMALL INTESTINE
ENZYMES, 5222
ULTRASTRUCTURE, 5222

PERIHEPATITIS
DISEASES ASSOCIATED WITH, 6122*
SURGERY, 6122*

PERISTALSIS
SMALL INTESTINE
CHOLERA, 5601

PERITONEOSCOPY
ABDOMEN
TRAUMA, 5625*

PERITONEUM
PANCREATITIS
DIAGNOSIS, 6047*

PERITONEUM (continued)
PATHOLOGY
FATTY ACIDS, 5209*
SCHISTOSOMIASIS, 6555

PERITONITIS
ABDOMEN
TRAUMA, 5625*
SMALL INTESTINE
SURGERY, 5892

PERMEABILITY
SMALL INTESTINE
CHELATING AGENTS, 5226
SURFACTANTS, 5226
VITAMIN A, 5237*
STOMACH
ACIDITY, 5265
CIRCULATION, 5265
ENDOTOXIN, 5207*
OXYGEN, 5265
PEPTIC ULCER, 5803
RADIATION, 5803

PEROXIDATION
LIPIDS
RADIOPROTECTIVE AGENTS, 5578
SMALL INTESTINE, 5578
LIVER
CARBON TETRACHLORIDE, 5419*

PEUTZ-JEGHERS SYNDROME
COMPLICATIONS
SURGERY, 5879

PHAGOCYTOSIS
LEUKOCYTE
APPENDICITIS, 5944
LEUKOCYTES
HEPATITIS, VIRAL, 6254
LIVER
CALCIUM, 5391*
TRAUMA, 6125*

PHENOBARBITAL
ALCOHOL
METABOLISM, 6315*
BILE
LIPIDS, 5442
SULFOBROMOPHTHALEIN, 5411*
BILE ACIDS
METABOLISM, 5480
BILIARY
ATRESIA, 6227
LIVER
RIBOSOMES, 5479
ULTRASTRUCTURE, 5373*
LIVER INJURY
HYDROCARBONS, HALOGENATED, 5416*
PROTEIN
SYNTHESIS, 5550
SMALL INTESTINE
ENZYMES, 5562*
LYSOSOMES, 5562*

PHOSPHATASE, ACID
PARASITES AND PARASITIC DISEASE
ULTRASTRUCTURE, 6563

PHOSPHATASE, ALKALINE
BILE
CHOLESTASIS, 6387*
CIRCADIAN RHYTHM, 6387*
BLOOD
DIETARY FACTORS, 5432
DRUG EFFECTS ON, 5541
ISOENZYMES, 6113*
LIVER DISEASE, 6113*
ISOENZYMES
NEOPLASMS, MALIGNANT, 6115*
LIVER
NEOPLASMS, MALIGNANT, 6115*
LIVER DISEASE
ISOENZYMES, 6094*
PANCREAS
NEOPLASMS, MALIGNANT, 6024*

PHOSPHATE
METABOLISM
LIVER, 5475
SALIVARY GLANDS
SECRETION, 5611

PHOSPHODIESTERASE
STOMACH
STARVATION, 5301*

PHOSPHOLIPASE
PANCREATITIS
DRUG TREATMENT, 6046*

PHOSPHOLIPID
BILE, 5429
EXCRETION
BILE, 5424
BILE ACIDS, 5424
LIVER
CHEMICAL COMPOSITION, 5540
GALACTOSAMINE, 5503
LIVER INJURY
CARBON TETRACHLORIDE, 5419*
METABOLISM
CALCIUM, 5350
LIVER, 5545
PANCREAS, 5350
SYNTHESIS
COENZYMES, 5238*
LIVER, 5472

PHOSPHORYLATION, OXIDATIVE
LIVER
ANTIBIOTICS, 5443
DRUG EFFECTS ON, 5413*
FOOD ADDITIVES, 5450
JAUNDICE, OBSTRUCTIVE, 5380*, 5389*
SHOCK, 5413*
PANCREAS
PANCREATITIS, 6046*

PHOTOTHERAPY
BILIRUBIN
METABOLISM, 5461

PHYSICAL FACTORS
WATER
ABSORPTION, 6340*

PHYSICAL PROPERTIES
BLOOD
CIRRHOSIS, 6338*
HEPATITIS, CHRONIC, 6338*
SMALL INTESTINE
DISACCHARIDASE, 5582

PIGMENTATION
COPPER
CIRRHOSIS, BILIARY, 6344*

PLASMA
SEE BLOOD
PROTEIN
HISTAMINE, 5311*
METABOLISM, 5293
STOMACH, 5303*, 5311*

PNEUMATOSIS
COLON, 5945
LARGE INTESTINE
ETIOLOGY, 5996
OXYGEN, 5929*
RESPIRATORY SYSTEM, 5996
THERAPY, 5929*
SMALL INTESTINE
ASCITES, 5895

POLYPHENOLS
AMINO ACIDS
ABSORPTION, 5261

POLYPS
SEE ALSO NEOPLASMS, BENIGN
APPENDIX
RADIOLOGY, 5958
CECUM
BLEEDING, 5995
PATHOLOGY, 5995
COLON
ENDOSCOPY, 5980
PATHOLOGY, 5947
PRECANCER, 5943, 5947
REVIEWS, 6001
SURGERY, 5946, 5980
LARGE INTESTINE
BACTERIA, 5928*
EPIDEMIOLOGY, 5990
FAMILIAL FACTORS, 5928*, 5931*
SURGERY, 5775
RECTUM
PRECANCER, 5943, 5964
STOMACH
DIAGNOSIS, 5659
DISEASES ASSOCIATED WITH, 5780
MENETRIERS DISEASE, 5780
NEOPLASMS, MALIGNANT, 5780
SURGERY, 5775

PORPHYRIA
LIVER
PATHOLOGY, 6157
REVIEWS, 6174
THERAPY
REVIEWS, 6164

PORTACAVAL SHUNT
COMPLICATIONS
DIET, 6353
HEPATORENAL SYNDROME, 6086*
LIVER
HYPERTROPHY, 5364*
LIVER INJURY
ENDOTOXIN, 5397*
SEQUELAE
PEPTIC ULCER, 5288*
PREVENTION, 5288*
SULFOBROMOPHTHALEIN
KINETICS, 6334*
TECHNIQUES, 5467

PORTAL HYPERTENSION
ANGIOGRAPHY, 6347*, 6360
CIRCULATION, 6341*
CIRRHOSIS
ASCITES, 6351
SURGERY, 6372
SURVIVAL, 6372
DISEASES ASSOCIATED WITH, 6192
FIBRINOGEN
SYNTHESIS, 6343*
LAPAROSCOPY, 5694
LIVER
CIRCULATION, 6343*
LIVER INJURY
ENDOTOXIN, 5397*
SURGERY
REVIEWS, 6364
VASOPRESSIN
CIRCULATION, 6348*

POTASSIUM
ABSORPTION
CELIAC DISEASE, 5910*
SMALL INTESTINE
SUCRASE, 5243*
TRANSPORT
GLUCOSE, 5257
JEJUNUM, 5257
SMALL INTESTINE, 5252*

PRECANCER
COLON
POLYPS, 5943, 5947
REVIEWS, 5956
CROHNS DISEASE
TRANSFORMATION, 6591
LIVER
NITROSAMINES, 5523
VITAMIN C, 5523
RECTUM
POLYPS, 5943, 5964
ULCERATIVE COLITIS
COMPLICATIONS, 6015*

PREGNANCY
CELIAC DISEASE
DIET, 5906*
CHOLESTASIS
REVIEWS, 6159
LIVER
CHOLESTASIS, 6108*
LIVER FUNCTION TESTS
REVIEWS, 6159

PRIMATES
AFLATOXIN
METABOLISM, 5524
CHENODEOXYCHOLIC ACID
TOXICITY, 5386*
CHOLELITHIASIS
SIMULATION, 5370*
LIVER
CIRCULATION, 5210*
DRUG METABOLISM, 5295
NEOPLASMS, MALIGNANT
BILIARY, 5483
SALIVARY GLANDS
DRUG METABOLISM, 5295
SMALL INTESTINE
DRUG METABOLISM, 5295
TRIGLYCERIDE
METABOLISM, 5376*
VITAMIN B12
DEFICIENCY, 5455
METABOLISM, 5455

PROGNOSIS
BILE DUCT
ATRESIA, 6211*
CHOLELITHIASIS
AGE FACTORS, 6422
HEPATITIS
NEONATE, 6211*
HEPATITIS, ACUTE
IMMUNOGLOBULINS, 6243*
HEPATITIS, CHRONIC, 6310
HEPATITIS, VIRAL, 6285
LIVER COMA
DRUG METABOLISM, 6078*
LIVER DISEASE, ALCOHOLIC
IMMUNOGLOBULINS, 6320
IMMUNOLOGY, 6314*
TRANSFERRIN, 6320
ULCERATIVE COLITIS, 6017

PROLAPSE
LIVER
HERNIA, 6166

PROSTAGLANDIN
BILE
SECRETION, 5381*
GLUCOSE
SYNTHESIS, 5452
SMALL INTESTINE
CYCLIC GUANOSINE MONOPHOSPHATE, 5575*
STOMACH
ACID SECRETION, 5381*
MOTILITY, 5281
SECRETION, 5800*

PROTEASE
PANCREATITIS
BLEEDING, 6064

PROTEIN
ABSORPTION
INTESTINE, 5266
NEONATE, 5266
BLOOD
CROHNS DISEASE, 6581*
LIVER DISEASE, 6137
SCHISTOSOMIASIS, 6569
ULCERATIVE COLITIS, 6581*
DEFICIENCY
CIRRHOSIS, 5473
LIVER, 5456
SMALL INTESTINE, 5598*
DUODENUM
ULTRASTRUCTURE, 5565*
IMMUNOLOGY
SMALL INTESTINE, 5600*
LIVER
HORMONE CONTROL, 5426
HYPOXIA, 5437
NEOPLASMS, MALIGNANT, 5460
TEMPERATURE, 5437
THYROID, 5426
METABOLISM
LIVER, 5519, 5542
PANCREAS
SECRETION, 5349
PLASMA
HISTAMINE, 5311*
METABOLISM, 5293
STOMACH, 5303*, 5311*
SECRETION
ALKALOIDS, 5374*
LIVER, 5374*
PANCREAS, 5341*, 5352
SMALL INTESTINE
LACTASE, 5573*
STOMACH
ACID SECRETION, 5313
LYMPH, 5303*
MALABSORPTION, 5737*
SYNTHESIS
ALCOHOL, 5372*
ANTIBIOTICS, 5343*
CYCLIC ADENOSINE MONOPHOSPHATE, 5495
LIVER, 5372*, 5478, 5479, 5488, 5495, 5522, 5550, 6139
PANCREAS, 5351
PHENOBARBITAL, 5550
SMALL INTESTINE, 5227
TRANSFUSION, 6139

PROTEIN-LOSING ENTEROPATHY
ENTERITIS
INFECTION, VIRUS, 5904*
IMMUNOGLOBULINS
DEFICIENCY, 5903*

PROTEINASE
GASTROINTESTINAL
VITAMIN B6, 5610

PROTHROMBIN
BLOOD
LIVER DISEASE, 6148

PROTOZOA
DRUG EFFECTS ON
ANTIBIOTICS, 6576

PSEUDOCYST
PANCREAS
ABSCESS, 6044

PSEUDOCYST (continued)
PANCREAS (continued)
BLEEDING, 6036, 6066
COMPLICATIONS, 6058, 6066
THERAPY, 6058

PSEUDOTUMOR
COLON
AMEBIASIS, 6007
LARGE INTESTINE
CHILDREN, 5870
LIVER
PATHOLOGY, 6100*
THERAPY, 6107*
RECTUM
RADIOLOGY, 5999
SMALL INTESTINE
CHILDREN, 5870

PSYCHOLOGICAL FACTORS
CONSTIPATION, 5994

PSYCHOTHERAPY
ULCERATIVE COLITIS, 6017, 6018

PYLOROPLASTY
ESOPHAGUS
RADIOLOGY, 5836
PEPTIC ULCER, 5841
STOMACH
ACID SECRETION, 5312*
MORPHOLOGY, 5855
RADIOLOGY, 5836
VAGOTOMY, 5851

PYLORUS
SEE ALSO STOMACH
ANOMALY
PEPTIC ULCER, 5755
HYPERTROPHY
PATHOLOGY, 5779
STENOSIS
HYPERTROPHY, 5771, 5791
NEONATE, 5791
NEOPLASMS, MALIGNANT, 5771
PATHOLOGY, 5779

RADIATION
SMALL INTESTINE
NEOPLASMS, MALIGNANT, 5588
STOMACH
PERMEABILITY, 5803

RADIOIMMUNOASSAY
BILE ACIDS
TOLERANCE, 5616*
BLOOD
MOTILIN, 5273*
GASTRIN
BLOOD, 5685

RADIOISOTOPES
LIVER FUNCTION TESTS
HYPERTENSION, 6155

RADIOLOGY
APPENDIX
POLYPS, 5958

RADIOLOGY (continued)
BILE DUCT
DISEASE, 5674
BILIARY, 6392*
BILIARY DISEASE, 5678
CHOLEDOCHOLITHIASIS, 6446
COLITIS
ISCHEMIA, 5987
COLON
CYSTS, 5993
GLYCOGEN STORAGE DISEASE, 6204
NEOPLASMS, MALIGNANT, 5932*
TECHNIQUES, 5658, 5679
DEFECATION
CHILDREN, 5638*
CONSTIPATION, 5638*
HIRSCHSPRUNGS DISEASE, 5638*
DIVERTICULUM
LARGE INTESTINE, 6005
DUODENUM
DISEASES, 5667
DIVERTICULUM, 5863*
ESOPHAGUS
ATRESIA, 5722, 5733
PYLOROPLASTY, 5836
SCLERODERMA, 5650
VAGOTOMY, 5836
VOMITING, 5716
GALLBLADDER
MOTILITY, 6467
GALLBLADDER DISEASE, 5662, 5674, 5678, 6389*
GASTROINTESTINAL
FOREIGN BODIES, 6476*
TUBERCULOSIS, 5986
HIRSCHSPRUNGS DISEASE
NEONATE, 5623*
ILEUS, 5937*
IRRITABLE COLON, 6005
LACTASE DEFICIENCY
SMALL INTESTINE, 5920
LIVER
ABSCESS, 6132
NEOPLASMS, MALIGNANT, 5696
PANCREAS, 6392*
NEOPLASMS, MALIGNANT, 5689, 5693
PANCREATITIS, CHRONIC, 5693
RECTUM
COMPLICATIONS, 6009
NEOPLASMS, MALIGNANT, 5932*
PSEUDOTUMOR, 5999
SMALL INTESTINE
ALLERGY, 5888
IMMUNITY, 5888
NEOPLASMS, 5656
NEOPLASMS, MALIGNANT, 5683
TECHNIQUES, 5686
STOMACH
NEOPLASMS, MALIGNANT, 5661, 5740
PATHOLOGY, 5617*
PYLOROPLASTY, 5836
VAGOTOMY, 5836
VOMITING
DIAGNOSIS, 5716

RADIOPROTECTIVE AGENTS
LIPIDS
PEROXIDATION, 5578
SMALL INTESTINE
IRRADIATION, 5233

RADIOTHERAPY
ESOPHAGUS
NEOPLASMS, MALIGNANT, 5710, 5715
GASTROINTESTINAL
NEOPLASMS, MALIGNANT, 6499
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 5933*
LIVER
NEOPLASMS, MALIGNANT, 6196, 6199
PEPTIC ULCER, 5803
RECTUM
NEOPLASMS, MALIGNANT, 5941, 5942
STOMACH
NEOPLASMS, MALIGNANT, 5746, 5750

RECTUM
SEE ALSO LARGE INTESTINE
BIOPOTENTIALS
CARBENOXALONE, 5799*
DISEASES
EPIDEMIOLOGY, 5984
FISTULA
NEOPLASM, MALIGNANT, 5950
NEOPLASMS, MALIGNANT
DIAGNOSIS, 5981
DISEASES ASSOCIATED WITH, 6004
ENDOSCOPY, 5692
ISCHEMIA, 5965
RADIOLOGY, 5932*
RADIOTHERAPY, 5941, 5942
SURGERY, 5941, 5942, 5949, 5981
SURVIVAL, 6000
POLYPS
PRECANCER, 5943, 5964
PSEUDOTUMOR
RADIOLOGY, 5999
RADIOLOGY
COMPLICATIONS, 6009
SURGERY
SEQUELAE, 5952
ULTRASTRUCTURE
ENDOCRINE SYSTEM, 5228

REFLUX
ESOPHAGUS
STENOSIS, 5714
SURGERY, 5718
PEPTIC ULCER
ETIOLOGY, 5822
SURGERY, 5802*

REGIONAL ENTERITIS
DISEASES ASSOCIATED WITH
MALABSORPTION, 6586
DUODENUM
STENOSIS, 6592

REJECTION
LIVER
TRANSPLATATION, 5398*
SMALL INTESTINE
TRANSPLANTATION, 5577

RESPIRATORY SYSTEM
CIRRHOSIS
DISEASES ASSOCIATED WITH, 6153
DRUG EFFECTS ON, 6345*
HEPATITIS, VIRAL
DISEASES ASSOCIATED WITH, 6287

RESPIRATORY SYSTEM (continued)
LARGE INTESTINE
PNEUMATOSIS, 5996
STOMACH
NEOPLASMS, MALIGNANT, 5762
ULCERATIVE COLITIS
DISEASES ASSOCIATED WITH, 6012*

RETICULOENDOTHELIAL SYSTEM
LIVER
TRAUMA, 6125*

REVIEWS
ALCOHOLISM
CIRRHOSIS, 6326
IMMUNOLOGY, 6332
BILE
SECRETION, 5434
BILIARY
DYSKINESIA, 6426
BILIARY DISEASE
JAUNDICE, 6429
BILIRUBIN
METABOLISM, 5421, 6177
CHOLESTASIS
PREGNANCY, 6159
CIRRHOSIS, 6327, 6361
ASCITES, 6359
THERAPY, 6187, 6300
CIRRHOSIS, BILIARY
AUTOIMMUNITY, 6101*
COLON
ENDOSCOPY, 5698
POLYPS, 6001
PRECANCER, 5956
DIARRHEA
ETIOLOGY, 6509
DRUG METABOLISM
LIVER, 5440
ENTERITIS
ETIOLOGY, 6504
ESOPHAGUS
VARICES, 6336*
FATTY LIVER
ALCOHOLISM, 6327
THERAPY, 6163
GASTROINTESTINAL
BLEEDING, 6508
DYSKINESIA, 6514
MOTILITY, 5279
GASTROINTESTINAL DISEASE
IMMUNOLOGY, 6506
HEMOCHROMATOSIS
ALCOHOLISM, 6329
THERAPY, 6164
HEPATITIS, CHRONIC, 6303, 6305
AUTOIMMUNITY, 6101*
DIAGNOSIS, 6307
DRUG TREATMENT, 6302
THERAPY, 6187, 6300, 6307
HEPATITIS, NONVIRAL, 6327
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 6277
AUTOIMMUNITY, 6101*
COMPLICATIONS, 6255
SEQUELAE, 6265
THERAPY, 6257

REVIEWS (continued)
INFLAMMATORY BOWEL DISEASE
EPIDEMIOLOGY, 6520
IMMUNOLOGY, 6520
JAUNDICE
DIAGNOSIS, 6427
LARGE INTESTINE
DIVERTICULITIS, 5968
DIVERTICULUM, 5968
LIVER
CHOLESTASIS, 6427
METABOLISM, 5463, 6134
PORPHYRIA, 6174
LIVER COMA
THERAPY, 6367
LIVER DISEASE, 6134
ALCOHOLISM, 6331
CHILDREN, 6208
DISEASES ASSOCIATED WITH, 6130
METABOLISM, 6104*
NEONATE, 6208
SCANNING, SCINTILLATION, 5666
THERAPY, 6141, 6173
LIVER DISEASE, ALCOHOLIC, 6321, 6327, 6331
IMMUNOLOGY, 6312*, 6328
SURGERY, 6322
LIVER FUNCTION TESTS
PREGNANCY, 6159
LIVER INJURY
ANALGESICS, 6231
MALABSORPTION
DIAGNOSIS, 5691
PANCREAS
NEOPLASMS, 6515
PANCREAS DISEASE
ANGIOGRAPHY, 6383*
SCANNING, SCINTILLATION, 5666
PORPHYRIA
THERAPY, 6164
PORTAL HYPERTENSION
SURGERY, 6364
SMALL INTESTINE
ENDOCRINE SYSTEM, 5220
STOMACH
MOTILITY, 5282
NEOPLASMS, MALIGNANT, 5796
SECRETION, 5335
ZOLLINGER-ELLISON SYNDROME
NEOPLASMS, 6515

REYES SYNDROME
ASPIRIN, 6093*
EPIDEMIOLOGY, 6093*
INFECTION, VIRUS, 6093*, 6197
LIVER
PATHOLOGY, 6095*, 6110*, 6233
VIRUS, 6110*
LIVER FUNCTION TESTS, 6110*

RIBOSOMES
LIVER
CARCINOGENS, 5458
PHENOBARBITAL, 5479
REGENERATION, 5469

RISK FACTORS
APPENDICITIS
SURVIVAL, 5979

RISK FACTORS (continued)
CIRRHOSIS
ALCOHOL, 6339*
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 6242*

RUPTURE
CECUM
NEOPLASMS, MALIGNANT, 5998
CYSTS
ECHINOCOCCOSIS, 6570
DUODENUM
TRAUMA, 5890
ESOPHAGUS
THERAPY, 5726
LIVER
AUTOIMMUNITY, 6198
CORTICOSTEROIDS, 6198
DISEASES ASSOCIATED WITH, 6198

SALIVA
ENZYMES
STOMACH DISEASE, 5742

SALIVARY GLANDS
ACETYLCHOLINE
SYNTHESIS, 5291
DRUG METABOLISM
ANTINEOPLASTIC AGENTS, 5295
PRIMATES, 5295
NEOPLASMS
ULTRASTRUCTURE, 6495
NEOPLASMS, MALIGNANT
DISEASES ASSOCIATED WITH, 6535
SECRETION
CROHNS DISEASE, 6582*
PHOSPHATE, 5611
TEMPERATURE, 5611

SALMONELLOSIS
ANTIBIOTICS, 6525
CARRIER STATE, 6512
CARRIER STATE, 6502
DRUG TREATMENT
ANTIBACTERIALS, 6540
ANTIBIOTICS, 6540
FECES
IMMUNOLOGY, 6518
FOOD
IMMUNOLOGY, 6518
TRANSMISSION
CHILDREN, 6538
VACCINE
CARRIER STATE, 6512
IMMUNITY, 6510, 6511, 6529
WATER
IMMUNOLOGY, 6518

SARCOIDOSIS
LIVER
NEOPLASMS, 6106*

SCANNING, SCINTILLATION
BILIARY DISEASE
TECHNIQUES, 5396*
CIRRHOSIS, 6368
GASTROINTESTINAL DISEASE, 5680
ILEUM
ISCHEMIA, 5605

SCANNING, SCINTILLATION (continued)
JAUNDICE
NEONATE, 5621*
JEJUNUM
INTUSSUSCEPTION, 5635*
LIVER
ABSCESS, 5627*
BILIARY DISEASE, 5634*
CIRCULATION, 6350
NEOPLASMS, MALIGNANT, 5630*, 6083*
REGENERATION, 5465
LIVER DISEASE
REVIEWS, 5666
TECHNIQUES, 5668, 5699
LIVER FUNCTION TESTS
SULFOBROMOPHTHALEIN, 6183
MECKELS DIVERTICULUM
BLEEDING, 5687
PANCREAS
NEOPLASMS, MALIGNANT, 5665
PANCREAS DISEASE, 5673
REVIEWS, 5666
PANCREATITIS, 5665
PAPILLA OF VATER, 5676

SCHISTOSOMIASIS
BLOOD
COPPER, 6003
IMMUNOGLOBULINS, 6550*
PROTEIN, 6569
ZINC, 6003
COLON
PERFORATION, 5983
IMMUNOLOGY, 6560
LIVER
FIBROSIS, 6003
PERITONEUM, 6555

SCLERODERMA
ESOPHAGUS
ENDOSCOPY, 5650
RADIOLOGY, 5650
GASTROINTESTINAL
SURGERY, 6541

SCLEROSIS
LIVER
DISEASES ASSOCIATED WITH, 6184

SEASONAL FACTORS
APPENDICITIS, 5957

SECRETIN
BILE
SECRETION, 5445
LIVER
CIRCULATION, 5445
PANCREAS
NEOPLASMS, MALIGNANT, 5690
SECRETION, 5347
PANCREAS DISEASE
DIAGNOSIS, 5681
PANCREAS FUNCTION TESTS
CHOLELITHIASIS, 6391*
PANCREATITIS, CHRONIC
DIAGNOSIS, 5647
PANCREOZYMIN, 5647
STOMACH
ACID SECRETION, 5322

SECRETION
AMYLASE
CALCIUM, 5350
PANCREAS, 5350
BILE
ENTERECTOMY, 5425
PEPTIC ULCER, 5820
PROSTAGLANDIN, 5381*
REVIEWS, 5434
SECRETIN, 5445
BILE ACIDS
LIVER, 5353*
BILIARY
DRUG EFFECTS ON, 6178
LIVER DISEASE, 6178
CHYMOTRYPSIN
PANCREAS, 5341*
COLON
BILE ACIDS, 5572*
GASTRIN
ALCOHOL, 5348
GASTRITIS, ATROPHIC
ANTICHOLINERGIC AGENTS, 5741
STEROID, 5741
VITAMINS, 5741
GASTROINTESTINAL
CHOLERETIC AGENTS, 5289
JEJUNUM
OBSTRUCTION, 5580
PANCREAS
ALCOHOL, 5287*, 5348
ANEMIA, PERNICIOUS, 6029*
ANTIBIOTICS, 5343*
CALCIUM, 5337*
CALCULI, 6027*
CHOLECYSTOKININ, 5574*
CHOLELITHIASIS, 6391*
CHOLINERGIC AGENTS, 5337*
DRUG EFFECTS ON, 5338*, 6178
ENTERECTOMY, 5425
GASTRECTOMY, 5806
GASTRITIS, ATROPHIC, 6029*
HEMOCHROMATOSIS, 6041
HORMONE EFFECTS ON, 5335
IMMUNOGLOBULINS, 6026*
LIVER DISEASE, 6178
MALNUTRITION, 6481*
NERVOUS CONTROL, 5287*, 5345
PROTEIN, 5349
SECRETIN, 5347
SYMPATHOMIMETIC AGENTS, 5347
ZOLLINGER-ELLISON SYNDROME, 6029*
PANCREATITIS, CHRONIC
CALCIFICATION, 6054*
PANETH CELL
DRUG EFFECTS ON, 5576*
STARVATION, 5576*
PEPSIN
PEPTIC ULCER, 5848
STOMACH, 5848
PEPTIDES
SMALL INTESTINE, 5198*
PROTEIN
ALKALOIDS, 5374*
LIVER, 5374*
PANCREAS, 5341*, 5352
SALIVARY GLANDS
CROHNS DISEASE, 6582*

SECRETION (continued)
SALIVARY GLANDS (continued)
PHOSPHATE, 5611
TEMPERATURE, 5611
SMALL INTESTINE
ENZYMES, 5583
STOMACH
ADRENERGIC BLOCKING AGENTS, 5798
AGE FACTORS, 5330
ANTICHOLINERGIC AGENTS, 5812, 5829
ANTIEMETICS, 5738*
ANTISECRETORY AGENTS, 5329
ANTRUM, 5335
ASPIRIN, 5798
BILE, 5305*
CHOLECYSTITIS, 6489
COLITIS, 6489
GASTRIN, 5331, 5335
GASTRITIS, 6489
H2 RECEPTOR ANTAGONISTS, 5332
HEIDENHAIN POUCH, 5305*
HISTAMINE, 5809
HORMONE EFFECTS ON, 5333
IMMUNOTHERAPY, 5829
PENTAGASTRIN, 5327
PEPTIC ULCER, 5800*, 5801*, 5809, 5812, 5819, 5829, 6489
PROSTAGLANDIN, 5800*
REVIEWS, 5335
STRESS, 5336
THYROID, 5744
TRANQUILIZERS, 5801*
ULCER, 5299*
VAGOTOMY, 5327, 5824
STOMACH DISEASE
DIAGNOSIS, 5675

SEDATIVES
ABDOMEN
PAIN, 5882
DUODENUM
DYSKINESIA, 5882

SEPSIS
LIVER
CIRCULATION, 5404*
OXYGEN, 5404*
LIVER COMA
ETIOLOGY, 6112*

SEROLOGICAL DIAGNOSIS
HEPATITIS, VIRAL
ANTIGEN, AUSTRALIA, 6276
RECURRENCE, 6235*

SEROLOGY
ESCHERICHIA COLI
DIARRHEA, 6486*

SEROTONIN
BLOOD
DUMPING SYNDROME, 5739*
PANCREATITIS, 6074
STOMACH
MOTILITY, 5284

SERUM
SEE BLOOD

SEX FACTORS
LIVER
DRUG METABOLISM, 5558

SHIGELLOSIS
DIARRHEA
ETIOLOGY, 6537
TOXIN, 6537
VACCINE
IMMUNITY, 6531

SHOCK
BLEEDING
DRUG TREATMENT, 5413*
CARBOHYDRATE
METABOLISM, 5496
LIVER
PHOSPHORYLATION, OXIDATIVE, 5413*
ULTRASTRUCTURE, 5501
SMALL INTESTINE
TRANSPLANTATION, 5607

SHORT BOWEL SYNDROME
SIMULATION
ENZYMES, 5566*
SURGERY
TECHNIQUES, 5898

SHUNT
BILE DUCT
LIVER, 6109*

SHUNT, INTESTINAL
COMPLICATIONS
CHOLELITHIASIS, 6403*
UROGENITAL SYSTEM, 6403*
ILEUM
GROWTH FACTOR, 5224
HYPERTROPHY, 5224
OBESITY
CHOLELITHIASIS, 6116*
CIRRHOSIS, 6111*
COMPLICATIONS, 6056*
FATTY LIVER, 5570*, 6111*, 6116*
LIVER FUNCTION TESTS, 6116*
PANCREATITIS, 6056*
SURGERY, 5894, 5896, 5899, 5900, 6390*, 6403*
SEQUELAE
CHOLELITHIASIS, 6390*
VITAMIN B12
MALABSORPTION, 5899

SIALIC ACID
LIVER
CARBON TETRACHLORIDE, 5419*

SIGMOID
SEE ALSO LARGE INTESTINE
VOLVULUS
DISEASES ASSOCIATED WITH, 5967

SKIN
SEE ALSO INTEGUMENTARY SYSTEM

SMALL INTESTINE
SEE ALSO DUODENUM, JEJUNUM, ILEUM
SEE ALSO INTESTINE
ABDOMEN
IRRADIATION, 5581

SMALL INTESTINE (continued)
ABSORPTION
SUGAR, 5251*
AMEBIASIS
NECROSIS, 6575
AMINO ACIDS
ABSORPTION, 5253, 5261
BIOCHEMISTRY
CORTICOSTEROIDS, 5199*
BIOPOTENTIALS
CHOLECYSTECTOMY, 6418
FEEDING, 5259
HORMONE EFFECTS ON, 5259
BIOPSY
LIVER DISEASE, 5672
MALABSORPTION, 5672
PANCREAS DISEASE, 5672
CALCIUM
ABSORPTION, 5263
CARCINOIDS
CROHNS DISEASE, 5934*
CHOLERA
IMMUNOLOGY, 5602
CHOLESTEROL
SYNTHESIS, 5579
CHOLINE
ABSORPTION, 5238*
CHYME
NITROGEN, 5290
CIRCULATION
TRANSPLANTATION, 5711
CYCLIC GUANOSINE MONOPHOSPHATE
CHOLINERGIC AGENTS, 5575*
HORMONE EFFECTS ON, 5575*
PROSTAGLANDIN, 5575*
DISACCHARIDASE
DEFICIENCY, 5919
PHYSICAL PROPERTIES, 5582
DIVERTICULUM
BLIND LOOP SYNDROME, 5917
MALABSORPTION, 5917
PEPTIC ULCER, 5875
DRUG ABSORPTION
DYES, 5262
DRUG METABOLISM
ANTINEOPLASTIC AGENTS, 5295
PRIMATES, 5295
ENDOCRINE SYSTEM
MORPHOLOGY, 5198*
REVIEWS, 5220
ENDOSCOPY
TECHNIQUES, 5684
ENZYMES
CELIAC DISEASE, 5222
CHOLECYSTOKININ, 5583
CIRCADIAN RHYTHM, 5561*
GASTRIN, 5583
PERFUSION, 5222
PHENOBARBITAL, 5562*
SECRETION, 5583
FISTULA
CROHNS DISEASE, 6593
SURGERY, 6395*
GLUCOSE
TRANSPORT, 5250*
GLYCOPROTEINS
SYNTHESIS, 5563*
HERNIA
SURGERY, 5876

SMALL INTESTINE (continued)
HYPERTROPHY
ENTERECTOMY, 5571*
NERVOUS SYSTEM, 5571*
IMMUNOGLOBULINS
AGE FACTORS, 5604
GNOTOBIOSIS, 5604
IMMUNOLOGY
CHOLERA, 5601
ION TRANSPORT
LAXATIVES, 5269*
SURFACTANTS, 5269*
IRRADIATION
ISCHEMIA, 5233
MORPHOLOGY, 5233
RADIOPROTECTIVE AGENTS, 5233
VITAMIN B12, 5598*
ISCHEMIA
PATHOLOGY, 5219
KINETICS, CELL
IRRADIATION, 5592*
LACTATION, 5200*
LACTASE
DIETARY FACTORS, 5573*
LACTOSE, 5573*
PROTEIN, 5573*
LACTASE DEFICIENCY
LACTOSE INTOLERANCE, 5920
RADIOLOGY, 5920
LIPIDS
ALBUMIN, 5600*
METABOLISM, 5568*
PEROXIDATION, 5578
SYNTHESIS, 5569*
LYMPHOCYTE
IMMUNOLOGY, 5594*
LYSOSOMES
PHENOBARBITAL, 5562*
MICROORGANISMS
BILIARY DISEASE, 6411
MOITLITY
LAXATIVES, 5269*
SURFACTANTS, 5269*
MORPHOLOGY
CHELATING AGENTS, 5226
CORTICOSTEROIDS, 5199*
DRUG EFFECTS ON, 5227
ENTEROKINASE, 5214*
ENZYMES, 5214*
MITOCHONDRIA, 5202*
MITOSIS, 5202*
SUCRASE, 5214*
SURFACTANTS, 5226
VAGOTOMY, 5221
MOTILITY
ACETYLCHOLINE, 5267*
ADRENERGIC AGENTS, 5267*
H2 RECEPTOR ANTAGONISTS, 5564*
HISTAMINE, 5274*, 5564*
MORPHINE, 5267*
NERVOUS SYSTEM, 5274*
MUCOPOLYSACCHARIDE
WHIPPLES DISEASE, 5915
NECROSIS
DISEASES ASSOCIATED WITH, 6552
NEOPLASMS
CHILDREN, 5870
RADIOLOGY, 5656

SMALL INTESTINE (continued)
NEOPLASMS, BENIGN
ANGIOGRAPHY, 5655
NEOPLASMS, MALIGNANT
ANGIOGRAPHY, 5655
COMPLICATIONS, 5878
IMMUNOLOGY, 5587
RADIATION, 5588
RADIOLOGY, 5883
NUCLEIC ACIDS
SYNTHESIS, 5234
NUCLEOTIDES
ABSORPTION, 5238*
OBSTRUCTION, 5881
DIVERTICULUM, 5869
ENDOMETRIOSIS, 5862*
THERAPY, 5884
PANETH CELL
DRUG EFFECTS ON, 5197*
PATHOLOGY
ANTIMETABOLITES, 5294
BLIND LOOP SYNDROME, 5205*
DERMATITIS HERPETIFORMIS, 5905*
FATTY ACIDS, 5209*
IRRADIATION, 5592*
WHIPPLES DISEASE, 5915
PEPTIDASE
DRUG EFFECTS ON, 5197*
PEPTIDES
ABSORPTION, 5244*
SECRETION, 5198*
TRANSPORT, 5240*
PERISTALSIS
CHOLERA, 5601
PERMEABILITY
CHELATING AGENTS, 5226
SURFACTANTS, 5226
VITAMIN A, 5237*
PNEUMATOSIS
ASCITES, 5895
POTASSIUM
TRANSPORT, 5252*
PROTEIN
DEFICIENCY, 5598*
IMMUNOLOGY, 5600*
SYNTHESIS, 5227
PSEUDOTUMOR
CHILDREN, 5870
RADIOLOGY
ALLERGY, 5888
IMMUNITY, 5888
TECHNIQUES, 5686
REGENERATION
CELIAC DISEASE, 5913
MALABSORPTION, 5913
STASIS
ENZYMES, 5566*
SUCRASE
POTASSIUM, 5243*
SODIUM, 5243*
SURGERY
COMPLICATIONS, 5892
INFECTION, BACTERIA, 5892
PERITONITIS, 5892
TISSUE CULTURE
CELIAC DISEASE, 5907*
TRANSPLANTATION
NECROSIS, 5577
SMALL INTESTINE (continued)
TRANSPLANTATION (continued)
REJECTION, 5577
SHOCK, 5607
TRANSPORT
SURGERY, 5264
TUBERCULOSIS, 5887
ULTRASTRUCTURE
CELIAC DISEASE, 5222
PEPTIDES, 5567*
PERFUSION, 5222
VITAMIN A
DEFICIENCY, 5237*, 5598*
VITAMIN B1
TRANSPORT, 5247*
VITAMIN B12
ABSORPTION, 5598*
VITAMIN D
ABSORPTION, 5387*

SOCIOECONOMIC FACTORS
LARGE INTESTINE
NEOPLASMS, MALIGNANT, 5927*
STOMACH
NEOPLASMS, MALIGNANT, 5927*

SODIUM
CHOLERA
TOXIN, 5256
ILEUM
MOTILITY, 5283
SMALL INTESTINE
SUCRASE, 5243*
TRANSPORT
CHOLERA, 5256
ILEUM, 5242*, 5256

SONOGRAPHY
SEE ECHOGRAPHY

SPECIES DIFFERENCES
LIVER
PERFUSION, 5492

SPHINCTER
BILE
NERVOUS CONTROL, 5517
PRESSURE STUDIES, 5517
ESOPHAGUS
PRESSURE STUDIES, 5306*, 5713

SPLEEN
KIDNEY
HYPERPLASIA, 5511
LIVER
REGENERATION, 5511
PATHOLOGY
LIPIDS, 5521

STARCHES
BLOOD
ALBUMIN, 5456
HYDROLYSIS
ENTERITIS, 5865*
PANCREATITIS, CHRONIC, 5865*

STARVATION
LIVER
CIRCULATION, 5404*

STARVATION (continued)
LIVER (continued)
MITOSIS, 5532
OXYGEN, 5404*
PANETH CELL
SECRETION, 5576*
STOMACH
ADENYL CYCLASE, 5301*
PHOSPHODIESTERASE, 5301*

STASIS
SMALL INTESTINE
ENZYMES, 5566*

STEATOSIS
SEE FATTY LIVER

STENOSIS
COLON
PANCREAITITS, 6061
DUODENUM
REGIONAL ENTERITIS, 6592
ESOPHAGUS
DISEASES ASSOCIATED WITH, 5730
REFLUX, 5714
SURGERY, 5714, 5720, 5730
PAPILLA OF VATER
DIAGNOSIS, 5640
PYLORUS
HYPERTROPHY, 5771, 5791
NEONATE, 5791
NEOPLASMS, MALIGNANT, 5771
PATHOLOGY, 5779

STEROID
CHOLELITHIASIS
DRUG-INDUCED, 5544
GASTRITIS, ATROPHIC
SECRETION, 5741
HEPATITIS, CHRONIC
CHILDREN, 6291*
LIVER
COMA, 6342*
METABOLISM
LIVER, 5390*
STOMACH
ACID SECRETION, 5649
TRANSPORT
LIVER, 5520
URINE
CIRRHOSIS, 6355

STOMACH, 5306*
SEE ALSO ANTRUM, PYLORUS
ABSORPTION
ASPIRIN, 5258
BILE ACIDS, 5255
CARBENOXALONE, 5258
ACID SECRETION
ACIDITY, 5310*
ALCOHOL, 5287*, 5302*
ANTISECRETORY AGENTS, 5315
ANTRECTOMY, 5312*
CALCITONIN, 5769
CALCIUM, 5307*, 5310*
CHOLERA, 5601
DRUG EFFECTS ON, 5312*, 5842
FEEDING, 5313
GASTRIN, 5321, 5322

STOMACH (continued)
ACID SECRETION (continued)
H2 RECEPTOR ANTAGONISTS, 5309*, 5324
HEPATECTOMY, 5297*
HISTAMINE, 5298*, 5309*, 5324, 5328, 5734*
HORMONE CONTROL, 5296*
INTESTINE, 5325
KIDNEY TRANSPLANTATION, 5751
MAGNESIUM, 5307*
NERVOUS CONTROL, 5287*
PANCREAS, 5325
PANCREATITIS, CHRONIC, 6059
PARASYMPATHOMIMETIC AGENTS, 5298*
PEPTIC ULCER, 5807
PROSTAGLANDIN, 5381*
PROTEIN, 5313
PYLOROPLASTY, 5312*
SECRETIN, 5322
STEROID, 5649
VAGOTOMY, 5298*, 5312*, 5734*, 5849
ACIDITY
GASTRECTOMY, 5825
PEPTIC ULCER, 5825
ADENYL CYCLASE
PENTAGASTRIN, 5301*
STARVATION, 5301*
ARTERY
ANEURYSM, 6542
BIOPOTENTIALS
ASPIRIN, 5258
CARBENOXALONE, 5258, 5799*
CHOLECYSTECTOMY, 6418
PEPTIC ULCER, 5260
BLEEDING
DIAGNOSIS, 5660
DRUG-INDUCED, 5660
EPIDEMIOLOGY, 5660
HYPOTHERMIA, 5794
SURGERY, 6500
TRACER STUDY, 5633*
CHEMICAL BURNS
ENDOSCOPY, 5754
CHOLECYSTOKININ
GASTRIN, 5321
CHYME
NITROGEN, 5290
CIRCULATION
ADRENERGIC AGENTS, 5597*
ALCOHOL, 5590*
ASPIRIN, 5590*
BILE ACIDS, 5590*
CORTICOSTEROIDS, 5317
DRUG EFFECTS ON, 5300*
VAGOTOMY, 5300*, 5326
VASOCONSTRICTORS, 5597*
CYCLIC ADENOSINE MONOPHOSPHATE
ALCOHOL, 5302*
DIVERTICULUM
DISEASES ASSOCIATED WITH, 5835
PEPTIC ULCER, 5875
ENDOCRINE SYSTEM
AGE FACTORS, 5334
GASTROINTESTINAL DISEASE, 5216*
ULTRASTRUCTURE, 5216*
ENDOSCOPY
TECHNIQUES, 5684

STOMACH (continued)
ENZYMES
STRESS, 5336
EROSIONS
ADRENERGIC BLOCKING AGENTS, 5798
ASPIRIN, 5798
BILE ACIDS, 5255
BLEEDING, 5794
ENDOSCOPY, 5785
HYPERPLASIA, 5785
PREVENTION, 5255
STRESS, 5318
FATTY ACIDS
DEFICIENCY, 5281
FISTULA
COLON, 5768
FOREIGN BODIES
DIAGNOSIS, 5765
PEPTIC ULCER, 5813
THERAPY, 5765
GALLBLADDER
FISTULA, 6442
GASTRIN
DUODENITIS, 5748
GASTRITIS, 5748
HYPERCHLORHYDRIA, 5748
IMMUNOLOGY, 5331
PEPTIC ULCER, 5748
SECRETION, 5331
ION TRANSPORT
PEPTIC ULCER, 5260
LYMPH
PROTEIN, 5303*
MALABSORPTION
PROTEIN, 5737*
SURGERY, 5737*
METASTASES
DISEASES ASSOCIATED WITH, 5735*
MORPHOLOGY
ENDOCRINE SYSTEM, 5225
ENDOTOXIN, 5207*
GASTRIN, 5225
INTRINSIC FACTOR, 5213*
PYLOROPLASTY, 5855
MOTILITY
ALKALOIDS, 5284
ANTIEMETICS, 5738*
CALCITONIN, 5769
CHOLECYSTOKININ, 5271*
CHOLINESTERASE, 5272*
DRUG EFFECTS ON, 5842
DUODENUM, 5275*
HYPERCHLORHYDRIA, 5769
MARKER STUDIES, 5275*
NERVOUS CONTROL, 5272*
OSMOTIC PRESSURE, 5275*
PEPTIC ULCER, 5823
PROSTAGLANDIN, 5281
REVIEWS, 5282
SEROTONIN, 5284
TRACER STUDY, 5752, 5823
NEOPLASMS, BENIGN
BIOPSY, 5615*
DIAGNOSIS, 5766
ENDOSCOPY, 5615*
SURGERY, 5766, 5773, 5777
ULTRASTRUCTURE, 5781
NEOPLASMS, MALIGNANT, 5789
ALBUMIN, 5770

STOMACH (continued)
NEOPLASMS, MALIGNANT (continued)
CALCIFICATION, 5778
CHEMOTHERAPY, 6484*
CIRCULATION, 5757
COMPLICATIONS, 5783
DIAGNOSIS, 5651, 5659, 5709, 5756, 6484*
ENDOSCOPY, 5619*, 5641, 5651, 5740, 5763
ENZYMES, 5767
FOREIGN BODIES, 5783
GASTRECTOMY, 5745
IMMUNOGLOBULINS, 5795
MARKER STUDIES, 5619*
NITROSAMINES, 5316
PATHOLOGY, 5736*, 5753, 5757, 5788, 5948
RADIOLOGY, 5661, 5740
RADIOTHERAPY, 5746, 5750
RECURRENCE, 5792
RESPIRATORY SYSTEM, 5762
REVIEWS, 5796
SIMULATION, 5316
SOCIOECONOMIC FACTORS, 5927*
SURGERY, 5750, 5782, 5786, 5792
SURVIVAL, 5763
THERAPY, 5753
PANCREATITIS, CHRONIC
CALCIFICATION, 6059
PATHOLOGY
ANALGESICS, 5308*
ANTIINFLAMMATORY AGENTS, 5308*
ASPIRIN, 5308*
RADIOLOGY, 5617*
STRESS, 5232
THYROID, 5744
VAGOTOMY, 5232
PEPSIN
SECRETION, 5848
PERMEABILITY
ACIDITY, 5265
CIRCULATION, 5265
ENDOTOXIN, 5207*
OXYGEN, 5265
PEPTIC ULCER, 5803
RADIATION, 5803
PHOSPHODIESTERASE
STARVATION, 5301*
PLASMA
PROTEIN, 5303*, 5311*
POLYPS
DIAGNOSIS, 5659
DISEASES ASSOCIATED WITH, 5780
MENETRIERS DISEASE, 5780
NEOPLASMS, MALIGNANT, 5780
SURGERY, 5775
RADIOLOGY
PYLOROPLASTY, 5836
VAGOTOMY, 5836
SECRETION
ADRENERGIC BLOCKING AGENTS, 5798
AGE FACTORS, 5330
ANTICHOLINERGIC AGENTS, 5812, 5829
ANTIEMETICS, 5738*
ANTISECRETORY AGENTS, 5329
ANTRUM, 5335
ASPIRIN, 5798
BILE, 5305*

STOMACH (continued)
SECRETION (continued)
CHOLECYSTITIS, 6489
COLITIS, 6489
GASTRIN, 5335
GASTRITIS, 6489
H2 RECEPTOR ANTAGONISTS, 5332
HEIDENHAIN POUCH, 5305*
HISTAMINE, 5809
HORMONE EFFECTS ON, 5333
IMMUNOTHERAPY, 5829
PENTAGASTRIN, 5327
PEPTIC ULCER, 5800*, 5801*, 5809, 5812, 5819, 5829, 6489
PROSTAGLANDIN, 5800*
REVIEWS, 5335
STRESS, 5336
THYROID, 5744
TRANQUILIZERS, 5801*
VAGOTOMY, 5327, 5824
SURGERY
VITAMIN B12, 5759
TELANGIECTASIA
CIRRHOSIS, 6358
ULCER
BLEEDING, 5797
COMPLICATIONS, 5790, 5797
CORTICOSTEROIDS, 5755
DIAGNOSIS, 5827
DISEASES ASSOCIATED WITH, 5835
DRUG EFFECTS ON, 5842
DRUG TREATMENT, 5819
ENDOSCOPY, 5764
FISTULA, 5790
PREVENTION, 5823
RECURRENCE, 5797
SECRETION, 5299*
STRESS, 5299*, 5787
SURGERY, 5845
ULTRASTRUCTURE
AGE FACTORS, 5334
VAGOTOMY
ANTRECTOMY, 5851
PYLOROPLASTY, 5851
TECHNIQUES, 5314
VARICES
DISEASES ASSOCIATED WITH, 6035
PANCREAS DISEASE, 6035
PANCREATITIS, 6035
VOLVULUS
BLEEDING, 5793

STOMACH DISEASE
COLON
FISTULA, 5790
DRUG-INDUCED
CORTICOSTEROIDS, 5776
SALIVA
ENZYMES, 5742
SECRETION
DIAGNOSIS, 5675
SYPHILIS, 5784

STRANGULATION
HERNIA, HIATUS
SURGERY, 5708

STRESS
STOMACH
ENZYMES, 5336
EROSIONS, 5318
PATHOLOGY, 5232
SECRETION, 5336
ULCER, 5299*, 5787
ULCER
ETIOLOGY, 5299*

STRICTURE
BILIARY
DILATATION, 6470

SUCRASE
SEE ALSO DISACCHARIDASE
SMALL INTESTINE
MORPHOLOGY, 5214*
POTASSIUM, 5243*
SODIUM, 5243*

SUCROSE
BLOOD
ALBUMIN, 5456
CHOLERA
THERAPY, 6478*
DIARRHEA
THERAPY, 6478*

SUGAR
SMALL INTESTINE
ABSORPTION, 5251*
TRANSPORT
MALNUTRITION, 6485*
MICROORGANISMS, 6485*

SULFHYDRYL
OXIDATION
LIVER, 5464

SULFOBROMOPHTHALEIN
BILE
EXCRETION, 5410*, 5411*
PHENOBARBITAL, 5411*
BILIARY DISEASE
LIVER FUNCTION TESTS, 6182
GLUTATHIONE
BINDING, 5410*
KINETICS
CIRRHOSIS, 6334*
LIVER DISEASE, 5637*
PORTACAVAL SHUNT, 6334*
LIVER DISEASE
DIAGNOSIS, 5642
LIVER FUNCTION TESTS, 6182
LIVER FUNCTION TESTS
SCANNING, SCINTILLATION, 6183
TECHNIQUES, 5637*
METABOLISM
LIVER, 5360*
TEMPERATURE, 5360*

SURFACTANTS
JEJUNUM
TRANSPORT, 5248*
SMALL INTESTINE
ION TRANSPORT, 5269*
MOTILITY, 5269*
MORPHOLOGY, 5226
PERMEABILITY, 5226

SYMPATHOMIMETIC AGENTS
LIVER
GLUCONEOGENESIS, 5484
PANCREAS
SECRETION, 5347

SYPHILIS
STOMACH DISEASE, 5784

TELANGIECTASIA
STOMACH
CIRRHOSIS, 6358

TEMPERATURE
BILIRUBIN
METABOLISM, 5461
LIVER
PROTEIN, 5437
PANCREATITIS
DIAGNOSIS, 5648
SALIVARY GLANDS
SECRETION, 5611
SULFOBROMOPHTHALEIN
METABOLISM, 5360*

TERPENES
PEPTIC ULCER
DRUG TREATMENT, 5843

THYROID
CHOLESTEROL
METABOLISM, 5379*
LARGE INTESTINE
DISEASE, 5960
LIVER
PROTEIN, 5426
PANCREATITIS
COMPLICATIONS, 5339*
STOMACH
PATHOLOGY, 5744
SECRETION, 5744

TISSUE CULTURE
LIVER
HEPATITIS, VIRAL, 6279
REGENERATION, 5405*
ULTRASTRUCTURE, 5449
PANCREAS
NEOPLASMS, 5593*
SMALL INTESTINE
CELIAC DISEASE, 5907*

TOLERANCE TEST
DISACCHARIDE
MALABSORPTION, 5670

TOXICITY
CHENODEOXYCHOLIC ACID
PRIMATES, 5386*
MERCURY
LIVER, 5395*
VITAMIN A
LIVER, 5355*

TOXIN
CHOLERA
SODIUM, 5256
SHIGELLOSIS
DIARRHEA, 6537

TOXOPLASMOSIS
BLOOD
IMMUNOGLOBULINS, 6550*

TRACE ELEMENT
BLOOD
CHOLECYSTITIS, 6412
HEPATITIS, VIRAL
BALANCE STUDIES, 6234*
CHILDREN, 6234*

TRANQUILIZERS
STOMACH
SECRETION, 5801*

TRANSAMINASE
BLOOD
DIETARY FACTORS, 5432
ENVIRONMENTAL FACTORS, 5441
GENETIC FACTORS, 5441

TRANSFERRIN
LIVER DISEASE, ALCOHOLIC
DIAGNOSIS, 6320
PROGNOSIS, 6320

TRANSFORMATION
ANUS
NEOPLASMS, BENIGN, 5976
CROHNS DISEASE
PRECANCER, 6591

TRANSFUSION
HEPATITIS, VIRAL
ETIOLOGY, 6266
PREVENTION, 6242*
PROTEIN
SYNTHESIS, 6139

TRANSPLANTATION
LIVER
ENZYMES, 5447
ISCHEMIA, 5537
LIPID STORAGE DISEASES, 6092*
LIVER DISEASE, ALCOHOLIC, 6323
MORPHOLOGY, 5447
NECROSIS, 6167
PATHOLOGY, 5398*
REJECTION, 5398*
TECHNIQUES, 5498, 5514
PANCREAS
CYTOLOGY, 5346
GENETICS, 5346
SMALL INTESTINE
CIRCULATION, 5711
NECROSIS, 5577
REJECTION, 5577
SHOCK, 5607

TRANSPORT
COLON
ADENYL CYCLASE, 5204*
GLUCOSE
GLYCOSIDES, 5250*
LACTASE DEFICIENCY, 5250*
SMALL INTESTINE, 5250*
JEJUNUM
BILE ACIDS, 5241*, 5248*

TRANSPORT (continued)
JEJUNUM (continued)
CHOLESTEROL, 5248*
SURFACTANTS, 5248*
PEPTIDES
SMALL INTESTINE, 5240*
POTASSIUM
GLUCOSE, 5257
JEJUNUM, 5257
SMALL INTESTINE, 5252*
SMALL INTESTINE
SURGERY, 5264
SODIUM
CHOLERA, 5256
ILEUM, 5242*, 5256
STEROID
LIVER, 5520
SUGAR
MALNUTRITION, 6485*
MICROORGANISMS, 6485*
VITAMIN B1
SMALL INTESTINE, 5247*

TRAUMA
ABDOMEN
PERITONEOSCOPY, 5625*
PERITONITIS, 5625*
DUODENUM
RUPTURE, 5890
LIVER
BLEEDING, 6129*
CHILDREN, 6193
DRAINAGE, 6203
HEPATECTOMY, 6152
OPSONIN, 6125*
PHAGOCYTOSIS, 6125*
RETICULOENDOTHELIAL SYSTEM, 6125*
SURGERY, 6129*
PANCREAS
IMMUNOGLOBULINS, 6026*

TREATMENT
SEE THERAPY

TRICHINOSIS
BLOOD
IMMUNOGLOBULINS, 6550*

TRIGLYCERIDE
BLOOD
FATTY ACIDS, 5435
LIVER DISEASE, 6123*
VITAMIN A, 5549
DUODENUM
FEEDING, 5286*
GASTRIN, 5286*
FATTY LIVER
ALCOHOL, 5376*
LIPIDS
METABOLISM, 5383*
LIVER
FATTY ACIDS, 5435
VITAMIN A, 5549
METABOLISM
ALCOHOL, 5376*
CHENODEOXYCHOLIC ACID, 6380*
KINETICS, 6380*
LIVER, 5376*

TRIGLYCERIDE (continued)
METABOLISM (continued)
LIVER DISEASE, 6123*
PRIMATES, 5376*
PANCREATITIS
ALCOHOL, 6049*

TRYPANOSOMIASIS
ESOPHAGUS
MOTILITY, 6547*

TRYPSIN
DUODENUM
GIARDIASIS, 6544*
PANCREAS
ANTINEOPLASTIC AGENTS, 5609
GIARDIASIS, 6544*
IRRADIATION, 5609

TUBERCULOSIS
GASTROINTESTINAL
COMPLICATIONS, 5986
RADIOLOGY, 5986
SMALL INTESTINE, 5887

ULCER
SEE ALSO DUODENUM, STOMACH
APPENDIX
DISEASES ASSOCIATED WITH, 5995
DIVERTICULUM
DUODENUM, 5886
DUODENAL
SURGERY, 5845
DUODENUM
DIAGNOSIS, 5827
ENDOSCOPY, 5826
STOMACH
BLEEDING, 5797
COMPLICATIONS, 5790, 5797
CORTICOSTEROIDS, 5755
DIAGNOSIS, 5827
DISEASES ASSOCIATED WITH, 5835
DRUG EFFECTS ON, 5842
DRUG TREATMENT, 5319
ENDOSCOPY, 5764
FISTULA, 5790
PREVENTION, 5323
RECURRENCE, 5797
SECRETION, 5299*
STRESS, 5299*, 5787
SURGERY, 5845
STRESS
ETIOLOGY, 5299*

ULCERATIVE COLITIS
SEE ALSO COLITIS
ANTIGEN, CARCINOEMBRYONIC, 6014*
BLOOD
PROTEIN, 6581*
COMPLICATIONS
HEMATOPOIETIC SYSTEM, 6021
MEGACOLON, 5973
NEOPLASMS, MALIGNANT, 6015*
PRECANCER, 6015*
SURGERY, 6020
DISEASES ASSOCIATED WITH
ALLERGY, 6011*
HEPATITIS, CHRONIC, 6525
LIVER DISEASE, 6012*
RESPIRATORY SYSTEM, 6012*

ULCERATIVE COLITIS (continued)
DRUG TREATMENT, 6020
ANTIBACTERIALS, 6019, 6023
ENDOSCOPY, 5622*
ILEOSTOMY
DIET, 6022
MICROORGANISMS, 6022
IMMUNOLOGY, 6580*
LARGE INTESTINE
MONOAMINOXIDASE, 6016
MOUTH, 6582*
NEOPLASMS, MALIGNANT
ANTIGEN, CARCINOEMBRYONIC, 6013*
PROGNOSIS, 6017
PSYCHOTHERAPY, 6017, 6018
SURGERY, 6523
THERAPY, 6505

UREA
LIVER
DIETARY FACTORS, 5354*
METABOLISM
HEPATITIS, VIRAL, 6283
SYNTHESIS
LIVER, 5354*, 5359*

URINE
ACIDITY
LIVER DISEASE, ALCOHOLIC, 6313*
AMINO ACIDS
PANCREATITIS, 6045*
ESTROGENS
LIVER DISEASE, ALCOHOLIC, 6317*
PEPSINOGEN
PEPTIC ULCER, 5819
STEROID
CIRRHOSIS, 6355

UROGENITAL SYSTEM
BILIARY DISEASE
SURGERY, 6414
CELIAC DISEASE
DISEASES ASSOCIATED WITH, 5923
LIVER DISEASE
DISEASES ASSOCIATED WITH, 6127*
PANCREATITIS
DISEASES ASSOCIATED WITH, 6067
SHUNT, INTESTINAL
COMPLICATIONS, 6403*

VACCINE
CHOLERA
IMMUNITY, 6527
DYSENTERY
IMMUNITY, 6530, 6532
ENTERITIS
ETIOLOGY, 6504
ESCHERICHIA COLI
ENTERITIS, 6528
HEPATITIS, VIRAL
COMPLICATIONS, 6241*
INTESTINE
INFECTION, BACTERIA, 6533
SALMONELLOSIS
CARRIER STATE, 6512
IMMUNITY, 6510, 6511, 6529
SHIGELLOSIS
IMMUNITY, 6531

VAGOTOMY
BLOOD
GASTRIN, 5804
DIARRHEA
THERAPY, 5859
ESOPHAGUS
RADIOLOGY, 5836
GALLBLADDER
MOTILITY, 5749
GLUCOSE
ABSORPTION, 5774
LIPIDS
ABSORPTION, 5774
PEPTIC ULCER, 5841
DRAINAGE, 5850
PREVENTION, 5288*
SEQUELAE, 5802*, 5815, 5824
SURGERY, 5817
TECHNIQUES, 5314, 5853
SMALL INTESTINE
MORPHOLOGY, 5221
STOMACH
ACID SECRETION, 5298*, 5312*, 5734*, 5849
ANTRECTOMY, 5851
CIRCULATION, 5300*, 5326
PATHOLOGY, 5232
PYLOROPLASTY, 5851
RADIOLOGY, 5836
SECRETION, 5327, 5824
TECHNIQUES, 5314

VARICES
ESOPHAGUS
CIRRHOSIS, 6336*
REVIEWS, 6336*
THERAPY, 6336*
STOMACH
DISEASES ASSOCIATED WITH, 6035
PANCREAS DISEASE, 6035
PANCREATITIS, 6035

VASOCONSTRICTORS
STOMACH
CIRCULATION, 5597*

VASOPRESSIN
LIVER
CIRCULATION, 5393*, 5415*, 5533
PORTAL HYPERTENSION
CIRCULATION, 6348*

VINYL CHLORIDE
LIVER INJURY
ECHOGRAPHY, 6224*

VIRUS
HEPATITIS
LIVER FUNCTION TESTS, 5499
PATHOLOGY, 5499
JEJUNUM
GASTROENTERITIS, 6536
LIVER
REYES SYNDROME, 6110*

VITAMIN A
BLOOD
TRIGLYCERIDE, 5549

VITAMIN A (continued)
DEFICIENCY
SMALL INTESTINE, 5237*, 5598*
LIVER
TRIGLYCERIDE, 5549
METABOLISM
LIVER, 5355*
SMALL INTESTINE
PERMEABILITY, 5237*
TOXICITY
LIVER, 5355*

VITAMIN B1
TRANSPORT
SMALL INTESTINE, 5247*

VITAMIN B2
DEFICIENCY
LIVER, 5525

VITAMIN B6
DEFICIENCY
GASTROINTESTINAL TRACT, 5610
JEJUNUM, 5239*
GASTROINTESTINAL
PROTEINASE, 5610
HEPATITIS, CHRONIC
DRUG TREATMENT, 6294*

VITAMIN B12
ABSORPTION
BLIND LOOP SYNDROME, 5902*
GASTRECTOMY, 5759
ILEOSTOMY, 5866*
JEJUNUM, 5902*
SMALL INTESTINE, 5598*
CIRRHOSIS
DRUG TREATMENT, 6352
DEFICIENCY
PRIMATES, 5455
LIVER DISEASE
DRUG TREATMENT, 6186
MALABSORPTION
CHILDREN, 5918
GROWTH RETARDATION, 5918
SHUNT, INTESTINAL, 5899
METABOLISM
PRIMATES, 5455
SMALL INTESTINE
IRRADIATION, 5598*
STOMACH
SURGERY, 5759

VITAMIN C
BILE
LIPIDS, 5371*
DEFICIENCY
CHOLELITHIASIS, 5544
LIVER
PRECANCER, 5523
PEPTIC ULCER
SURGERY, 5840

VITAMIN D
ABSORPTION
SMALL INTESTINE, 5387*
AMINO ACIDS
ABSORPTION, 5253
DEFICIENCY
ABSORPTION, 5253
METABOLISM
LIVER, 5387*

VITAMIN E
LIVER
ULTRASTRUCTURE, 5504

VITAMINS
GASTRITIS, ATROPHIC
SECRETION, 5741

VOLVULUS
CECUM
SURGERY, 5991
LARGE INTESTINE
OBSTRUCTION, 5988
SIGMOID
DISEASES ASSOCIATED WITH, 5967
STOMACH
BLEEDING, 5793

VOMITING
DIAGNOSIS
RADIOLOGY, 5716
ESOPHAGUS
RADIOLOGY, 5716

WATER
ABSORPTION
CIRRHOSIS, 6340*
PHYSICAL FACTORS, 6340*
EXCRETION
CIRRHOSIS, 6340*
JEJUNUM
ABSORPTION, 5246*
SALMONELLOSIS
IMMUNOLOGY, 6518

WHIPPLES DISEASE
ETIOLOGY
NEONATE, 5911
SMALL INTESTINE
MUCOPOLYSACCHARIDE, 5915
PATHOLOGY, 5915

WILSONS DISEASE
LIVER
PATHOLOGY, 6180

XYLOSE
ABSORPTION
CIRCULATION, 5925

ZIEVES SYNDROME
DISEASES ASSOCIATED WITH
INTEGUMENTARY SYSTEM, 6319

ZINC
BLOOD
SCHISTOSOMIASIS, 6003

ZOLLINGER-ELLISON SYNDROME
BLOOD
GASTRIN, 6483*, 6521
NEOPLASMS
REVIEWS, 6515
PANCREAS
NEOPLASMS, 5593*
SECRETION, 6029*
SURGERY, 6494

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